A study to assess correlation of information obtained from cardiac MRI viability scan which provides information on presence and transmural extent of myocardial infarction in a vascular territory with that derived from a combination of

- ECG (presence of an infarct),

- echocardiography (wall motion abnormality and left ventricle function) and

- conventional coronary angiography (involved coronary artery and degree of involvement in terms of percentage of stenosis),

in patients with chronic ischemic cardiomyopathy, in a tertiary care center in South India.

A dissertation submitted in partial fulfillment of MD Radiodiagnosis (Branch VIII) examination of the Tamil Nadu Dr. M.G.R Medical University, Chennai to be held in April 2015

CERTIFICATE

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Guide:

Dr. Elizabeth Joseph
Professor
Department of Radiodiagnosis
Christian Medical College
Vellore – 632004.

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Head of the Department:

Dr. Shyamkumar N K
Professor
Department of Radiodiagnosis
Christian Medical College
Vellore – 632004.

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Principal:

Dr. Alfred Job Daniel, D.Ortho, MS Ortho, DNB
Principal,
Christian Medical College & Hospital,
Vellore.

DECLARATION

I, Dr. Hannah L. Khiangte, hereby declare that this dissertation entitled "A study to assess correlation of information obtained from cardiac MRI viability scan which provides information on presence and transmural extent of myocardial infarction in a vascular territory with that derived from a combination of - ECG (presence of an infarct), echocardiography (wall motion abnormality and left ventricle function) and conventional coronary angiography (involved coronary artery and degree of involvement in terms of percentage of stenosis), in patients with chronic ischemic cardiomyopathy, in a tertiary care center in South India." is an original work done by me in partial fulfillment of the requirement for MD Radiodiagnosis (Branch VIII) Degree Examination of the Tamil Nadu Dr. M.G.R Medical University, Chennai to be held in April 2015.

Candidate:

Dr. Hannah L.Khiangte

Post Graduate Resident,

Department of Radio Diagnosis,

Christian Medical College & Hospital,

Vellore

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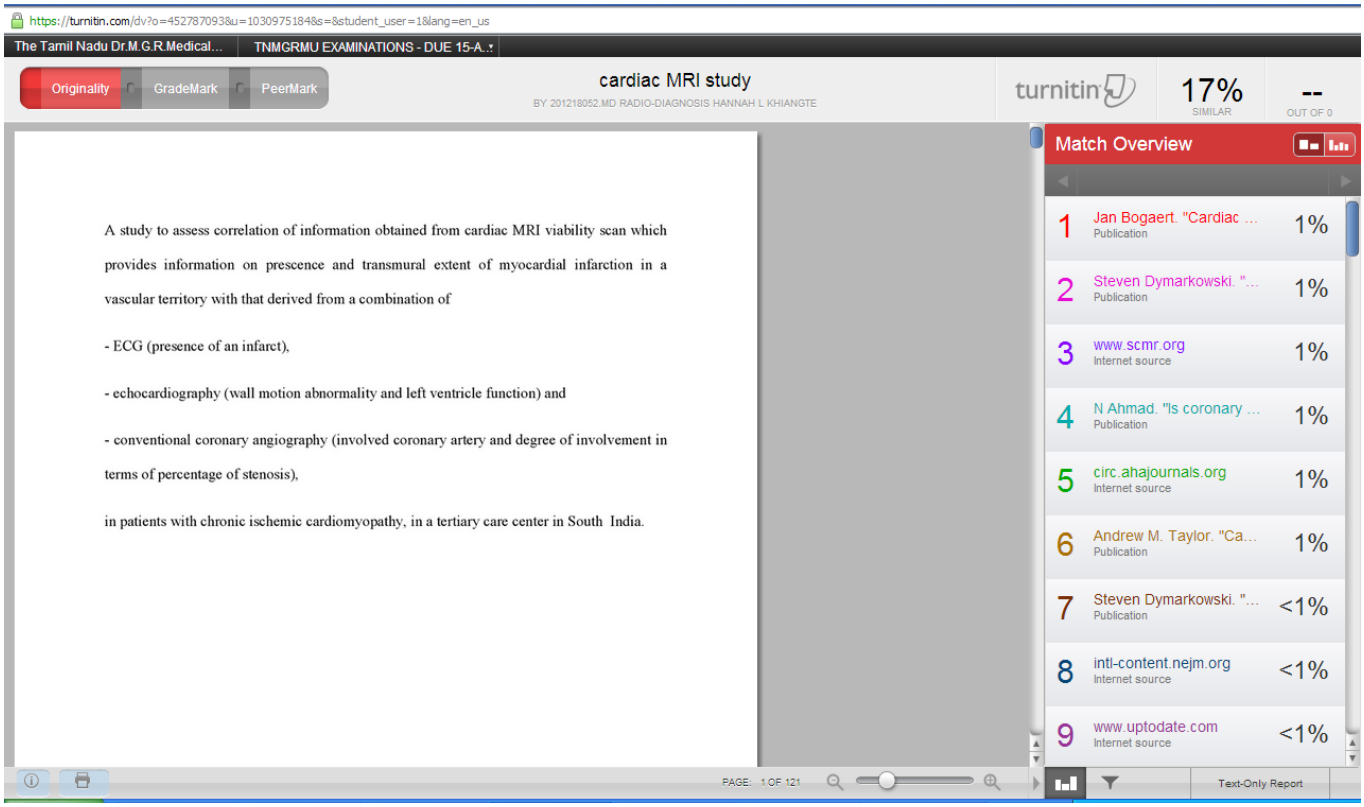

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ABSTRACT

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A study to assess correlation of information obtained from cardiac MRI viability scan which provides information on presence and transmural extent of myocardial infarction in a vascular territory with that derived from a combination of ECG (presence of an infarct), echocardiography (wall motion abnormality and left ventricle function) and conventional coronary angiography (involved coronary artery and degree of involvement in terms of percentage of stenosis), in patients with chronic ischemic cardiomyopathy, in a tertiary care center in South India.

DEPARTMENT: Radiodiagnosis

NAME OF CANDIDATE: Hannah L Khiangte

DEGREE AND SUBJECT: M.D Radiodiagnosis

NAME OF THE GUIDE:

Dr. Elizabeth Joseph

Professor

Department of Radiodiagnosis

Christian Medical College

OBJECTIVES:

To compare regions / segments of myocardial infarction, identified by cardiac MRI and that predicted by ECG, ECHO and catheter coronary angiogram in the same patient and study the

degree of correlation, to identify segments of regional wall motion abnormality (RWMA) using cardiac MRI and score the RWMA quantitatively, to compare RWMA detected and quantified by cardiac MRI with that determined by echocardiography in the same patients, to estimate left ventricular ejection fraction (LVEF) using cardiac MRI, and compare the results with echocardiographically determined LVEF by ECHO in the same patients.

METHODS:

Analytical and descriptive study to assess the correlation between semiquantitative method and cardiac MRI in the evaluation of myocardial infarct in ischemic heart disease. Consecutive patients with a clinical diagnosis of ischemic cardiomyopathy who have undergone an electrocardiography, echocardiography and coronary angiography to diagnose and evaluate the disease were recruited in the study. Reporting of electrocardiography, echocardiography and coronary angiogram was done by the cardiologist. Cardiac MRI viability scan performed was reported by the radiologist. Correlation of findings in terms of presence of infarct, wall motion abnormality, left ventricular ejection fraction, degree of vessel occlusion on catheter coronary angiography and extent of transmural delayed enhancement on cardiac MRI viability scan were analysed by scatter plot, Bland Altman plot and intra-class correlation coefficient. P-value of <0.05 was considered statistically significant.

RESULTS:

A total of segments from 20 patients were evaluated, of which 177 segments were diseased. We found that Cardiac MRI viability better depicts presence and extent of infarct. The same number of segments were identified as contractile dysfunction on echocardiography and eyeballing method on cine MRI images whereas fewer segments were identified by

myocardial wall thickness measurement method on MRI images. We concluded that wall thickness measurement method is poor in differentiating normal myocardial wall motion from mild to moderate wall motion abnormality. Regional wall motion abnormality (RWMA) assessed by eyeballing (MRI) and by myocardial wall thickness measurement (MRI) at end diastole and end systole both are able to distinctly distinguish myocardial segments into three categories of RWMA. Left ventricular function assessed as ejection fraction by echocardiography and MRI showed significant correlation, p value <0.05. Significant association between degree of coronary vessel occlusion and delayed enhancement of the corresponding myocardial segment was seen only in the Left anterior descending artery and its territory (p-value 0.05). We conclude that a protocol consisting of a combination of cardiac MR viability and coronary angiography will give all the information required in management of a patient.

Keywords: myocardial viability, delayed hyperenhancement, myocardial infarct, myocardial wall motion abnormality.

INTRODUCTION

Chronic ischemic cardiomyopathy is defined as impaired left ventricular function (ejection fraction =< 35 to 40 percent) that results from coronary artery disease. It is considered to be present in patients with heart failure who have had a myocardial infarction or have evidence of viable hibernating myocardium. Viable hibernating myocardium can be identified by markers of myocardial viability in regions with contractile dysfunction by various imaging techniques. Example: positron emission tomography (PET imaging), Thallium imaging and Dobutamine echocardiography). Two important pathologic mechanisms involved in the disease process are:

- One, loss of myocardial mass and contraction due to myocardial infarction which is irreversible. Recovery of function of such cannot be achieved by coronary revascularization since the tissue is infarcted.

- Two, loss of myocardial contractility due to reduced function of ischemic yet viable myocardium due to prior ischemic insult. This viable myocardium can be detected on imaging studies and can recover function after coronary revascularization.(1)

GLOBAL SCENARIO

Coronary artery disease is the single largest cause of death in developed countries and a leading cause of disease burden in developing countries. The prevalence of the disease is very variable in different regions because of multiple factors. 43% of all coronary vascular disease deaths are attributed to coronary heart disease. Globally coronary vascular deaths represent 30% of all deaths.(2)

INDIAN SCENARIO

The prevalence of ischemic heart disease in India is extensive, both in rural and urban population and is the leading cause of death in our country.(1) A comprehensive table of data of prevalence of ischemic heart disease at various locations from our country is presented in Appendix 1.(3) A point that needs specific mention from the data in the table is that, the mean age of presentation of coronary artery disease in our country is 57.5 years which is 7-11 years younger than when compared to western countries.(3) This illustrates the importance and significance of identification of the disease and tertiary prevention of the disease in addition to primary and secondary prevention in our country. Imaging plays an important role in the identification of treatable disease.

We now know that if we can identify irreversibly injured myocardium from those that are dysfunctional but viable, which means that they recover function, it will help us immensely and is therefore of utmost importance for the management of cardiac patients. Revascularization of an infarcted area by percutaneous coronary intervention or coronary artery bypass grafting is can only be justified by the fact that functional recovery will follow the intervention or if late outcome and patient well being can be improved after the procedure. (4)

Catheter coronary angiography is currently the gold standard of investigation to detect those that need surgical revascularization in our institution. Catheter coronary angiography is performed by the cardiologist in a state of the art “cath lab” in our institution. Results are classified as follows, lesions < 50% are minor; 50-69% are intermediate and >70% are

significant. Lesions >70% occlusion are considered needing revascularization. Lesions are measured by visual estimates or by QCA (quantitative coronary analysis) which is incorporated in the PHILIPS lab systems. For suspicious intermediate lesions (50-69%), revascularization is recommended only if one of the other non-invasive tests positive for reversible ischemia. The rest are on medical treatment and on routine follow up.

Cardiac MRI scan offers the greatest information from one single test. It allows the identification of a region of wall motion abnormality with no delayed enhancement which identifies a viable myocardium versus a region of wall motion abnormality with delayed enhancement which identifies an infarct in the myocardium. An infarct is seen on a delayed scan as subendocardial enhancement extending from the subendocardium to variable thickness of the myocardium depending on the severity of the disease.

The degree of transmurality of enhancement can prognosticate the outcome of a revascularization procedure which no other tests can do.(5) Localization of the arterial territory involved or diseased is possible by the 17 cardiac segments described by the American Heart Association. In addition, we can assess the left ventricular function on cardiac MRI.(6) Additionally stress MRI scan with adenosine stress test can further identify regions of inducible ischemia in the myocardium.

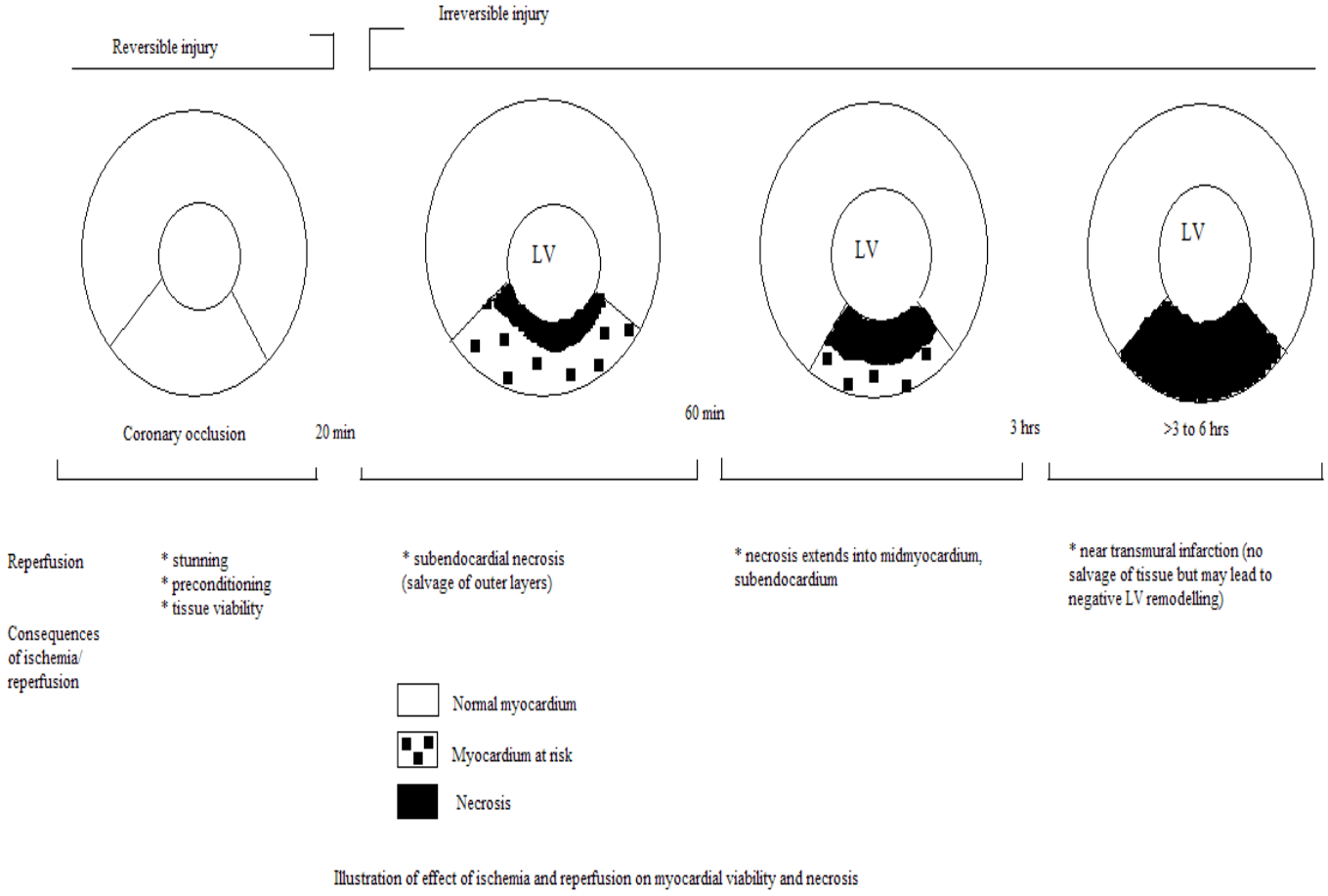


Fig. 1 - Illustration of effect of ischemia and reperfusion on myocardial viability and necrosis. Adapted from Clinical Cardiac MRI, Boegaert. (4)

In this study we have looked at the information obtained from a combination of ECG, ECHO and coronary angiography of a patient suspected to have ischemic heart disease and assessed whether this information agrees with information obtained from a cardiac MRI viability scan done and reported in the Radiology department in our institute. The agreement and correlation assessed are in terms of presence of infarct and wall motion abnormality. We have compared the left ventricular function obtained by ECHO and cardiac MRI and we have assessed the transmurality of enhancement of diseased segments. Depending on the percentage of wall thickness involvement we have predicted the extent to which the diseased segments may recover post revascularization.

AIMS AND OBJECTIVES

Primary objectives:

1. To compare regions / segments of myocardial infarction, identified by cardiac MRI and that predicted by ECG, ECHO and catheter coronary angiogram in the same patient and study the degree of correlation.

2. To identify segments of regional wall motion abnormality (RWMA) using cardiac MRI and score the RWMA quantitatively.

3. To compare RWMA detected and quantified by cardiac MRI with that determined by echocardiography in the same patients

4. To estimate left ventricular ejection fraction (LVEF) using cardiac MRI, and compare the results with echocardiographically determined LVEF by ECHO in the same patients.

Secondary Objectives:

1. To quantify the extent of transmural infarction in myocardial segments using delayed enhancement cardiac MRI, and predict the likelihood of improvement in the left ventricular contractile function after myocardial revascularization

REVIEW OF LITERATURE

Concept of myocardial viability

A cascade of events occur in the process of regional or global myocardial ischemia. This cascade starts with disturbed myocardial vascularization which is followed by decrease in diastolic and later systolic function. Finally, this is followed by electrocardiographic changes and the patient then suffers from typical symptomatic angina which prompts the electrocardiographic test in the first place.(4) The most important variable that influences outcome in patients suffering from coronary artery disease is the functionality or status of the left ventricle. Left ventricle dysfunction leads to poor quality of life and increases mortality among patients with coronary artery disease.

Viability of a myocardium is term that is used to describe a segment of the myocardium, which being supplied by a diseased coronary artery, is dysfunctional in terms of contractile function due to ischemia. This segment of myocardium with contractile dysfunction may have limited or absent scarring. The presence of a limited or absent scar means that this particular segment of the myocardium has potential for functional recovery following revascularization. This definition of viability is a prospective definition.

The term hibernation on the other hand is a retrospective definition. Hibernating myocardium is one that has shown evidence of functional recovery after intervention for revascularization.(7)

Hibernating myocardium is a state of adaptation. The myocardium displays diminished contractile function in keeping with or in order to match the chronically oxygen deprived cells. This phenomenon was recognized from clinical observation made more than 30 years ago by clinicians when they noticed that chronic myocardial dysfunction present before a bypass surgery did improve after revascularization.(8) So hibernating myocardium will improve or normalize after revascularization procedures.

Pathophysiology

Occlusion of the coronary arteries by pathology such as atherosclerosis, most commonly, results in interruption of the normal oxidative metabolism or normal cellular respiration of the myocardium. This is replaced by anaerobic metabolism. Anaerobic metabolism is a compensatory mechanism. This type of metabolism process results in accumulation of lactate and other acidic metabolites. Because of the resultant acidic environment the myocardium has very short lived capacity for the same. The final result is occurrence of acute myocardial ischemia, almost immediately, in the territory that is supplied by the occluded artery.

When acute myocardial ischemia occurs due to acute coronary occlusion, an ischemic cascade follows. The cascade consists of setting in of myocardial contractile dysfunction within seconds. With sustained ischemia, intracellular edema occurs within 20 to 30 minutes.

This is followed by irreversible injury to myocytes in 30 to 60 minutes and vascular endothelial cells within 60 to 90 minutes with cellular necrosis and apoptosis.(9)

In its physiological state, the subendocardial myocardium is at a constant state of higher energy demand than the rest of the myocardium.(4) The bearing of this is that necrosis of the myocardium progresses from the subendocardium to the subepicardium.(9) The time taken for necrosis to reach transmural extent is approximately 6 to 12 hours.(10) The end result of the entire cascade is necrosis and apoptosis, indicating myocardial infarction.

At the microscopic level, the site of necrosis is identified to be heavily loaded with markers of cellular inflammation such as macrophages. The macrophages engulf the necrotic cellular debris. In due time connective tissue then replaces the injured area converting it into a site of mature collagenous non contractile scar. Apart from necrosis that occurs from acute and severe ischemia a process of programmed cell death, apoptosis, also occurs in the myocardium. Apoptosis of myocytes requires at least a few days to manifest. This is the reason that at a very early stage of the cascade, the extent of injury can be underestimated at a microscopic level.

The first event of the cascade manifests in the myocardium as myocardial contractile dysfunction. In the absence of a continued ischemic insult this myocardial dysfunction recovers functionality completely. This is what is referred to as myocardial stunning. It is a result of the metabolic derangement that occurs in the post transient ischemia phase. It may take anywhere from days to weeks to recover the native contractile abilities. Multiple

episodes of ischemic events may lead to cumulative myocardial stunning which in turn leads to a hibernating myocardium and LV dysfunction. (11)

Rationale for viability imaging

In the management of patients with coronary artery disease and decreased left ventricular function resulting in poor quality of life and increased chances of sudden cardiac related death, it is important to consider three things. Firstly, primary prevention, which is the spread of awareness and health educating the population. This will prevent or reduce preventable risk factors occurring in a person such as smoking, hypertension, dyslipidemia, diabetes mellitus, obesity, physical inactivity and high stress. Secondly, secondary prevention, which is the management of risk factors that has already developed such as cessation of smoking, control of blood pressure, diet control and drug therapy for dyslipidemia and diabetes, regular exercise for weight management, antiplatelets and anticoagulants unless contraindicated, ACE inhibitors and beta blockers and influenza vaccination. Thirdly, tertiary prevention would be surgical management with revascularization in the presence of indication according to existing guidelines to reduce the morbidity caused by the already existing disease. Treatment of chronic ischemic heart disease is either medical or surgical. Patients undergo investigations like ECG, ECHO and coronary angiography as a part of work up for diagnosis of the disease.

It is recommended that viability of a myocardium be assessed in the work up process of the disease. Methods to identify a viable myocardium becomes very relevant with the

information at hand as it reflects the direction for management of patients with ischemic heart disease. With this knowledge that identification of irreversibly injured myocardium (infracted myocardium) from dysfunctional but viable and potentially salvageable myocardium is of crucial importance for the management of cardiac patients, availability of tests that differentiate one from the other become very important. Performing a revascularization procedure for a patient with ischemic heart disease with either percutaneous coronary intervention or coronary artery bypass grafting is considered justifiable only if we can be sure that functional recovery will follow the intervention. Or if it is predictable that late outcome and patient well being and improvement in day to day living activities can be improved.(4)

Evidence states that when a heart with viable myocardium is treated medically, it becomes a harbinger for further ischemic attacks that do not amount to being fatal but contribute significantly to an overall mortality which is much more than those on surgical management. Among the patient population having significant amount of viable myocardium, the mortality rate is > 4 fold greater annually when comparison was made between those treated medically and those who had had successful revascularization. Definite distinction between viable and nonviable myocardium which is dysfunctional therefore becomes necessary and crucial as it allows patients to benefit the success of revascularization if they are shown by investigations to have a viable myocardium. Any of the risks that procedures such as revascularizations are associated with can thereby be avoided in patients who have non viable myocardium and are unlikely to benefit from the surgery.

The assessment of myocardial viability

Only indirect methods are available to us to detect presence of living myocytes invivo in the clinical practice and research setting. Therefore clinically, viability is has been defined in various ways in accordance with the method used to indirectly identify the same. These are:

1) Recovery of contractile function following revascularization

2) Response to ionotropic stimulation (dobutamine or adenosine)

3) Presence of glucose metabolism (FDG PET imaging)

4) Presence of active cellular transport mechanisms (SPECT)

5) Presence of non scarred (absence of scar) myocardium on cardiac MRI viability imaging with delayed enhancement

Available modalities for assessment of viablilty:

Echocardiography

Echocardiography is one of the most valuable tool in the assessment of both regional and global cardiac dysfunction. It is the most widely used modality. It is also the most easily available technique. Stress echocardiography is a technique in which ionotropic stimulation of the myocardium allows us to identify functional changes that occur in the myocardium at rest (in the absence of ionotropic stimulation) and changes that occur at stress under ionotropic stimulation, simultaneously. The greatest disadvantage or limitation of

echocardiography other than the level of local expertise and the fact that it is observer dependant in nature is that of the presence of poor acoustic window. Patients with high body mass index and chest wall obesity, patients with lung disease and patients with prior heart surgery are poor candidates to echocardiography due to this drawback of poor acoustic window. This limitation can be overcome by transesophageal echocardiography however this technique may not be widely available both equipment wise and human resources wise. Another great limitation of echocardiography is the poor interface between the blood pool and the myocardium itself. This however can be overcome by tissue harmonic imaging. Contrast enhanced echocardiography is a technique which makes use of micro bubbles contrast. Is is one of the latest advantages in echocardiography. In addition to improving the contrast between the blood pool and the myocardium, micro bubbles may also play a role in myocardial perfusion assessment. It has been shown in certain studies to have clinical value in identifying perfusion defects in evaluation of acute myocardial infarction.(12)

Single Photon Emission Computed Tomography

Tl-201 and Tc-99m-sestamibi are the two most widely used radiopharmaceuticals.

Tl-201 Imaging:

This agent is potassium analog and enters cells via the sarcolemmal sodium potassium ATPase (adenosine triphosphatase) pump. A disrupted sacolemma indicates non viable myocardium as it is a marker that the cell is dead or infarcted. This tracer only enters cells with maintained or preserved sarcolemmal membrane, otherwise viable cells. Imaging is done immediately post induced stress either by exercise or pharmacological agents such as dobutamine or adenosine and at 3 to 4 hours later. Defects seen in the immediate images are

inducible ischemic regions. If they "fill in" in the delayed images, it indicates viability of that particular region. If they persists on the delayed images, the region is said to have a fixed defect. A fixed defect indicates one of 4 conditions- 1) reduction in the regional perfusion where the fixed defect occurs, 2) impairment in cellular integrity of the mycardium, 3) cell death or 4) scar tissue.(13) 22% of regions showing fixed defects are shown to "fill in" in late redistribution imaging indicating poorly perfused region with viable myocardium. (14)

Until recently, SPECT / PET was considered the gold standard in identifying viable myocardium. The reported sensitivity and specificity of SPECT is 86% and 74% respectively for coronary angiography proven coronary heart disease. (15)

A study by A Kapoor et al in 2006 revealed that cardiac MRI is superior to SPECT in identifying transmural infarct (non viable myocardial tissue) with sensitivity of cardiac MRI as 100% and sensitivity of SPECT as 79.3%(16)

Tc-99m- sestamibi Imaging:

Tc-99 sestamibi once injected binds to the mitochondria of the myocyte. The agent binds to the cell mitochondria as long as the mitochondrial matrix is intact and the transmembrane potential is negatively charged, which is indicative of a live or viable cell. The threshold of percent uptake is taken as 60%.(17) Findings of abnormality on sestamibi and Tl-201 imaging agrees quite well when the outcome of the imaging measured is in terms of predicting future recovery of the myocardium following procedures of revascularization.(10) Limitation of the examination is mainly in the spatial resolution which is poor. For this reason it is not possible to detect the varying extent of myocardial wall thickness involvement

in viability. The examination does not correctly delineate the area of infarction instead can only accurately point out if the uptake is above the threshold of 50% or 60% .

Positron Emission Tomography

Cardiac PET imaging is performed with FDG (18F-2-deoxy-2-fluoro-D-glucose) which is a glucose analogue. Following peripheral intravenous injection of the substrate, the pharmacokinetics of FDG follows the cellular pathway. Live or viable cells are therefore required for the FDG uptake to occur in the myocytes. It is incorporated into active cells via the same sarcolemmal protein as glucose and accumulates in the cells. The concentration of the tracer in the myocardium is made use of in estimating the rate at which glucose is utilized. An infarcted region of the myocardium will be metabolically inert and will not accumulate FDG within it. The resultant image is a region of low FDG concentration. Stunned or hibernating myocardium will show normal FDG uptake.

Limitations of PET is its high cost. It is not an easily available technique. Often it is non feasible to procure the tracer required for the test. The images obtained on PET also lack spatial resolution and is unable to determine exact percentage of wall thickness that is infarcted or the transmurality of the affectation.

MRI

Cardiac MRI is a versatile instrument to image the heart in the setting of coronary artery disease. Cardiac MRI has very high spatial resolution. It is able to distinguish transmural

variations in viability, accurately defining the extent of necrosis. There is therefore no suggestion that the technique will either underestimate or overestimate the extent of myocardial infarct. This is one of its strongest points as the spatial resolution in scintigraphy or PET imaging is relatively poor.

Gadolinium-DTPA is most commonly used as contrast agent in magnetic resonance imaging. It is an extracellular tracer with molecular size of 550 Da. Clearance of the substance from the body is by the kidneys. Healthy cells of the body exclude gadolinium chelated to DTPA. This contrast agent therefore is restricted to extravascular and interstitial spaces. Gadolinium-DTPA accumulates in infarcted cells. This is the loss of membrane integrity in the form of sarcolemmal break down in irreversibly injured or infarcted myocytes which allows the contrast agent to enter the extravascular intra cellular space. Further accumulation of the contrast agent is encouraged by delayed washout kinetics of the contrast due to suboptimal or poor venous drainage in these regions. Thus, increased signal enhancement of gadolinium-DTPA in delayed imaging is specific to myocardium that is irreversible injured.

Delayed imaging of the myocardium is done after several minutes of contrast injected via a peripheral line. The time lapse from the time of injection to imaging the myocardium can range from 10 to 20 min. Scar tissues or fibrosis will manifest as regions of hyperenhancement, i.e., high signal intensity on delayed enhancement MR imaging due to the contrast accumulation in the infarcted regions. Typically the hyperenhancement is subendocardial or transmural and in a coronary artery distribution. The hyperenhancement correlates on histopathology to fibrosis (scarring); it will not recover function post revascularization. This lead to the concept of "bright is dead". This is in reference to the high

signal intensity of the infarcted tissues. With this delayed imaging technique, infarcts upto 2 gram on average can be detected. (14)

In a landmark study Kim et al described the relationship of delayed enhancement in cardiac MRI to irreversible injury, infarct age (acute or chronic) and the contractile function of the myocardium in mongrel. Contrast enhancement of myocardium that was injured in acute infarction after induced severe but reversible ischemia was compared with contrast enhancement of myocardium in chronic infarction state. It was found that both acute and chronic myocardial infarcts enhance on the delayed contrast images and that the spatial extent of hyperenhancement is in keeping with the spatial extent of myocyte necrosis seen under microscope. On the other hand stunned myocardium did not enhance. The study established that myocardial scar show delayed enhancement and viable tissues do not show enhancement. the conclusion of the study was that MRI distinguishes between reversible and irreversible ischemic injury. They also concluded that this identification of reversible versus irreversible myocardium is likely to be independent of the wall motion and infarct age.(7)

Clinical implications from this study is that regions that do not hyperenhance are viable. The size and shape of the regions that show hyperenhancement are identical to tissues that show necrosis histopathologically and hence confirm irreversibility of the injury. This implies that we need not consider wall motion in the MRI definition of viability. Infact, according to the data of the study, myocardial wall thickening by cine MRI and viability by contrast MRI were dissociated in their study.

The assessment of myocardial viability which is the extent of infarction in a non invasive manner namely cardiac MRI in our study, is limited in that it only identifies tissues that may recover function after revascularization. It confirms the fact that there is a region of infarction which will not regain functionality and identifies areas of inducible ischemia. The test however does not depict coronary arteries and the state of disease in these arteries. It therefore needs to be combined with coronary angiography unless a coronary angiography is done as a part of the MR imaging. This technique is however in its early stages of development and not available with us. In the assessment ischemic heart disease in our study, it is not feasible to have histological verification clinically making the assessment an indirect one. The most commonly measured variable is the left ventricular ejection fraction.

Unlu et al, in a study, looked at left ventricular wall motion abnormality, perfusion and delayed contrast enhancement pattern in 24 patients with severely stenotic coronary artery, the severity being 70 to 99 percentage of vessel lumen occlusion. The result of the study showed that wall motion abnormalities, perfusion defects and delayed contrast enhancement were present in corresponding irrigation areas of the assessed stenotic vessels. They found a significant correlation in the cardiac MRI findings and the coronary angiogram findings. They concluded that the correlation was statistically significant and advised that a combined protocol of cardiac MRI and coronary angiography for the evaluation of ischemic heart disease is appropriate.(18)

In a study done to investigate the relationship between MRI determined infarct size (transmural extent) and long term functional improvement, the authors showed that the size

of the dysfunctional but normally enhancing region within a week of the acute event was the single best predictor of functional recovery 8 to 12 weeks post revascularization.(19)

We know now that the extent of wall thickness involvement (transmurality) of the delayed hyperenhancement predicts the probability of improved contraction after revascularization. Complete absence of hyperenhancement in segments of wall motion abnormality is known to completely recover function. Hyperenhancement of less than 25 percent of the myocardial thickness have increased likelihood of recovering function, and hyperenhancement of more than 50 percent of the myocardium are unlikely to recover function.(4,7)

When delayed imaging with gadolinium-DTPA and cine MRI assessements of contractility are used together in a protocol, we can establish the presence of normal or abnormal (stunned / infarcted) myocardium. Its extent can also be determined as follows: Based on cine MRI assessment alone, a segment with contractile dysfunction cannot be discriminated as stunned or infarcted. Viable tissue versus non viable tissue can be only be differentiated based on the difference in signal intensity after gadolinium-DTPA study.

Kim et al studied 50 patients with coronary artery disease and left ventricular dysfunction before the patients underwent revascularization procedure. They established the presence of 804 dysfunctional myocardial segments out of the 2093 segments that were studied. These dysfunctional segments also show varied extent of transmurality of delayed hyperenhancement on post gadolinium-DTPA scanning. They found that the likelihood of improvement in regional contractility after revascularization decreased progressively with the

transmural extent of hyperenhancement. In their results, contractility increased in 78% of segments with dysfunctional but no hyperenhancement before revascularization while only 1 out of 58 segments with more than 75% of wall thickness enhancements showed increased contractility. They concluded in the study that reversible myocardial dysfunction can definitely be identified by contrast enhanced MRI before a revascularization procedure.(20) Increased contractility in the myocardium post revascularization relates clinically to improvement in the left ventricular ejection fraction and overall improvement in day to day activities, lowered morbidity and increased survival.

A recent metaanalysis states that the annual mortality rate in patients with dysfunctional myocardium undergoing revascularization is more than twice as great in those without significant viability (7.7 percent) when compared to those with viable myocardium (3.2 percent). It also states that the perioperative mortality rate is increased by 10% in the absence of viability. (21)

In a study of 144 patients Bernhard et al reported that the three year prognosis was significantly worse among patients (with non dysfunctional myocardial segments which were viable) medically treated, when compared to those who did not have viable myocardium. They drew a conclusion that dysfunctional myocardium with preserved viability as shown by cardiac MRI viability scan is an independent predictor of mortality in patients suffering from ischemic heart disease.(22)

With increasing technological advances in cardiac MRI, there is place for cardiac MRI to be a one stop shop for evaluation of patients with ischemic cardiomyopathy as it is non-invasive and provides high resolution images of the heart in any desired plane. Another advantage is that cardiac MRI it is without radiation. (6) Because it is radiation free, the clinician is not limited by number of scans that can be requested in the follow up of a patient who is on treatment.

In most tertiary centers, a variety of investigations are used to diagnose coronary artery disease, risk stratify patients and plan their clinical management. To assess the need for revascularization surgery, majority of high risk patients undergo coronary angiography directly however lower / intermediate risk patients undergo non invasive tests like exercise tolerance testing (ETT), stress echocardiography or single photon emission computed tomography (SPECT), in order to identify those most likely to require coronary revascularization. (23) In the future cardiac MRI will play an important role in this algorithm of investigations for all the reasons that has already been stated.

Technique of gadolinium-DTPA contrast enhanced MR imaging

An inversion recovery sequence is used to null the normal myocardium. This results in better contrast difference between the normal myocardium and the infarcted myocardium which is contrast enhanced. The time of inversion is optimized for each patient just before the image acquisition. It is visually selected to maximize both signal to noise ratio and contrast to noise ratio of the left ventricle myocardium. Typical values for inversion time are 175 to 250 msec. It varies from person to person. (24) Following the R wave of the ECG, a delay period is used

to ensure that imaging of the heart occurs at the time of diastole. This is ensured because at diastole the motion of the beating heart is minimum.

ESSENTIALS OF MRI AND SPECIFIC FEATURES OF CARDIAC MRI

The nuclear spin phenomenon, T1 and T2 relaxation

Hydrogen protons in our body associated with fat and water molecules are positively charged and spins about its axis acting like tiny magnets, they are randomly aligned such that their magnetic fields cancel each other out rather than sum up. In MRI examination, the patient is made to lie in a high strength static magnetic field. The external magnetic field aligns the spins of the human body, some along the direction of the external magnetic field and some in the opposite direction. The interaction with the external magnetic field also results in precession of the protons. The Larmor equation determines the precessional frequency as f = gyromagnetic ratio x main magnetic field. The gyromagnetic ratio is constant and is characteristic for each type of nuclei.

The number of spins aligned in the direction of the external magnetic field being in excess produces a net magnetization (protons aligned in the direction of the external magnetic field minus protons aligned in the direction opposite to the external magnetic field). The net magnetization is the resting state, the most favorable situation energetically.

Resonance is a property that allows efficient transfer of energy. The net magnetization is in a direction parallel to the external magnetic field which is to say it is in the longitudinal direction (z direction). A radiofrequency pulse of certain amount of energy is transferred to the protons via the principle of resonance (excitation) such that the net magnetization flips to a certain degree rotating away from the longitudinal direction. The extent of rotation depends on the strength and duration of the RF pulse. The RF pulse strength and duration can be controlled. A 90 degree RF pulse rotates the net magnetization into the transverse plane (xy plane, x direction being left- right direction, y direction being anterior- posterior direction). A 180 degree RF pulse rotates the net magnetization into the - z direction.

After excitation, the spins of the protons return to their resting state. The magnetic component along the magnetic field increases and the component along the transverse plane or - z direction decreases. T1 relaxation is the gradual recovery of the magnetic field component along the net magnetization. T2 relaxation is the gradual disappearance of magnetization in the transverse plane.(25)

Slice encoding, Phase encoding, frequency encoding

T1 and T2 relaxation in itself is not enough to construct an image. Spatial encoding is a phenomenon whereby magnetic field gradients are applied over a volume of interest and an image is constructed. The process is based on the Larmor equation. The MR signal is localized by applying gradients that produce controlled linear variations in the magnetic field. These gradients are used in slice selection (z), phase encoding (y) and frequency encoding (x).

Slice selection gradient determines the amount of tissue that is excited by the RF pulse. Phase encoding gradient is applied perpendicular to the slice selection gradient and after the initial excitation. Frequency encoding is also referred to as the readout gradient since MR signal is acquired during the frequency encoding. It is applied to the third perpendicular direction to slice the selection and phase the encoding gradients. The protons are encoded with different frequency depending on their locations.

k-space

The data obtained from the gradients are stored in a matrix referred to as the k-space. High signal information is represented in the center and low signal information near the periphery, in the edges. When combined, the image has maximum resolution and contrast. Fourier transformation is then used on this information in the k-space to reconstruct images.(26)

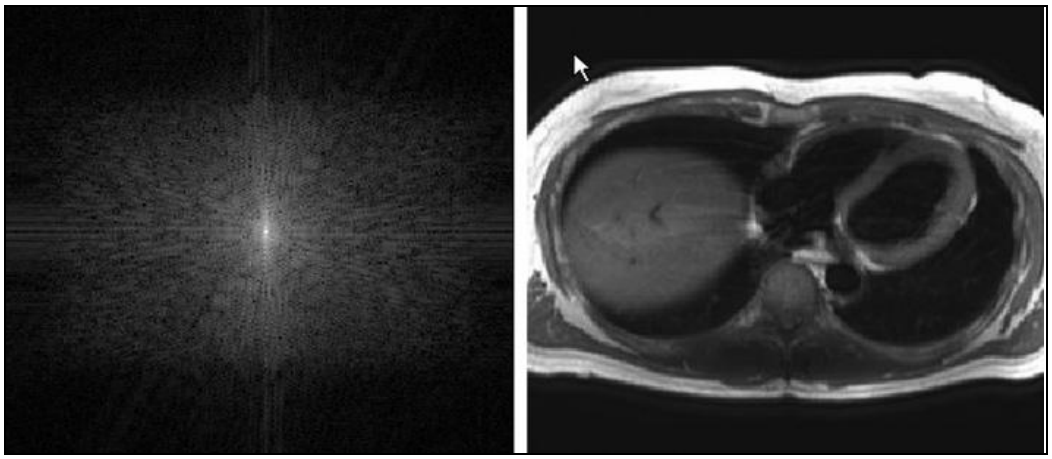


Fig. 2 - Information from both periphery and center of k-space gives maximum resolution and contrast.(4)

Spin echoes and Gradient echoes

Spin echo pulse sequences are now abandoned due to excessive motion artifacts. Fast spin echo / Turbo spin echo techniques are now employed which can acquire image in a single breath hold. They are made of a number of excitation pulses separated by a given time interval (TR). A period called the time of echo (TE) is the time between start of an RF pulse and the maximum in signal. It is repeated every TR (time of repetition) seconds. The TR is adjusted to coincide with one single R-R interval of the patient's ECG.

Steady State Free Precision

Synchronization with ECG during cardiac MRI is extremely important. SSFP sequence is a modification of gradient echo imaging wherein bright blood images are produced that contrasts the background myocardium resulting in a clear delineation between them. It is acquired by obtaining a steady state magnetization maintained between successive cycles. The high temporal resolution, excellent contrast between myocardium and blood within the chambers, and favorable SNR (signal to noise ratio) make it good for evaluation of wall motion and volumetric measurement.

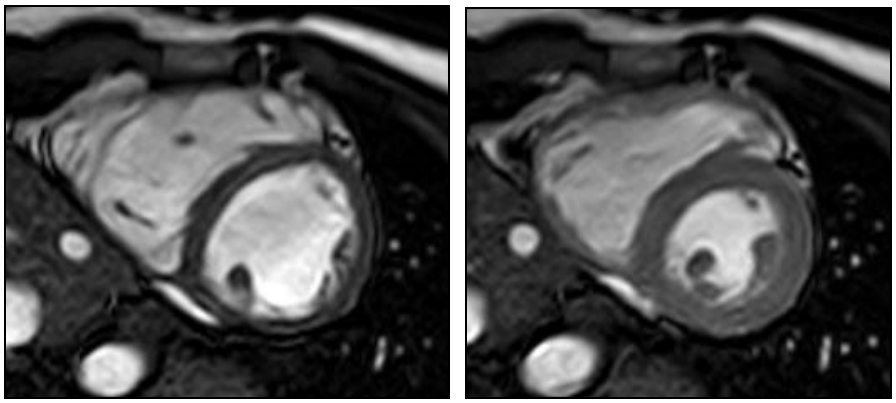


Fig. 3 – SSFP and GRE sequence in short axis at mid cavity level. SSFP shows better resolution and contrast. (4)

Phase sensitive inversion recovery sequence

Inversion recovery imaging is a sequence composed of a series of 180 degree pulses to invert the magnetization followed by a 90 degree pulse. The TR is between the 180 degree pulses. A time of inversion (TI) is chosen to selectively null a chosen tissue type signal. PSIR is a sequence wherein the normal myocardial tissue is suppressed / nulled. The TI for this sequence is variable and depends on several factors such as patients weight, contrast dose, renal function and time after injection. The final image shows a normal myocardium as dark and hyperenhanced myocardium as bright.(27)

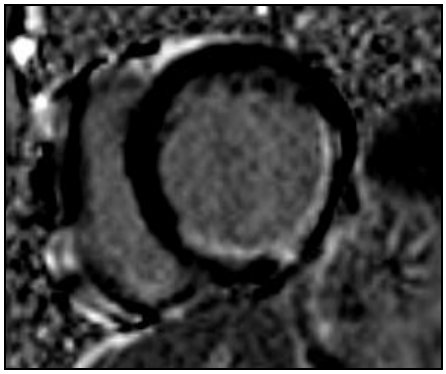


Fig. 4 – PSIR sequence showing delayed hyperenhancement in the inferior, inferoseptal and inferolateral segments at basal level

Advantages of cardiac MRI

1. Non invasive investigation with no ionizing radiation being involved.
2. Three dimensional modality makes it ideal for assessing structures in any plane, especially cardiac chambers.
3. Now considered the gold standard for cardiac viability, perfusion and stress testing
4. Ongoing research includes ways to provide information with regard to coronary arteries such as narrowing, type and degree of plaque. This leaves window open for more accurate diagnosis and better management planning of patients.

5. Pulmonary and systemic arteries of the chest are visualized in the study thereby providing a way to exclude other causes of chest pain such as pulmonary embolism or aortic dissection.
6. The 4D function of cardiac MRI gives accurate information of cardiac muscle and valve function and is not limited in visualization nor is it operator dependant as echocardiography.

Disadvantages of cardiac MRI

1. Relatively a lengthy examination, taking upto 30 to 60 minutes of machine time.
2. Patients suffering from claustrophobia, arrhythmias, obesity and those who are short of breath and have difficulty lying down may not be able to tolerate the examination.
3. Registration artifacts occur in the images resulting in blurred images in patients with tachycardia and irregular heart beats.
4. Coronary vessel assessment is suboptimal and it is difficult to assess the true degree of luminal narrowing especially in the small branches.
5. Implanted hardware such as pacemakers are contraindication to MRI examination.
6. The technology is not yet widely available and only few physicians are trained to perform and interpret these examinations.
7. Much clinical research is still needed to optimize the diagnostic algorithms in cardiac disease. Much awareness amongst cardiologist and adequate scientific literature with outcome analysis is required for fully comprehending the true value in diagnostic testing in specific clinical scenarios.

MRI ANATOMY OF THE HEART

Steady state free precession sequences best depict the cardiac anatomy. The heart is an intra thoracic organ which rests on the anterior aspect of the diaphragm in an oblique position with the lungs on either side and its apex in the left hemithorax.

Cardiac structures:

Right atrium: The right atrium is seen as the right heart border on the frontal chest radiograph, embryologically develops from the sinus venous and the primitive auricle. Characteristic features that distinguish the right atrium are 1) the venous component receiving the IVC and SVC in the posterior aspect and the coronary sinus in the inferior aspect, 2) the broad based, triangular shaped appendage, 3) a ridge called the crista terminalis which separates the venous part and the appendage, 4) limbus of the fossa ovalis on the septal aspect. Enlargement of the right atrium displaces the adjacent lung and enlargement of the appendage obliterates the retrosternal clear space.

Left atrium: Forms the upper posterior heart border lying just inferior to the carina and anterior in relation to the esophagus. Also forms from the sinus venosus and primitive auricle embryologically. Characteristic features that distinguish the left atrium are 1) the venous component receiving the four pulmonary veins at the corner of its posterior surface, 2) long finger like left atrial appendage that overlies the left atrioventricular groove and left

circumflex coronary artery, 3) absence of features that suggest right atrial morphology. When the left atrium is enlarged, it displaces the esophagus towards the posterior aspect. It also causes widening of the carina angle. Massive enlargement results in the left atrium forming the right heart border on the frontal chest radiograph. Enlargement of the appendage displaces the adjacent lung.

Atrial septum: The interatrial septum separates the left atrium from the right. It is best seen in the horizontal / transverse views and longitudinal / four chamber views. It is seen as a thin line separating the two atria except at the level of the foramen ovale which is very thin and therefore technically difficult to demonstrate, resulting in misinterpretation as an atrial septal defect which should be avoided.

Right ventricle: Pyramidal shaped chamber which rests on the diaphragm and forms the inferior and anterior heart border. The right ventricle does not from any part of the apex of the heart. Characteristic features that distinguish the right ventricles are 1) tendinous chord attachment of the tricuspid valve leaflets to the ventricular septum, 2) the moderator band (muscular band) containing a continuation of the right bundle branch and passing from the ventricular septum to the anterior wall, 3) the tricuspid valve and the pulmonary valves are separated by crista supraventricularis which is an infolding of the roof of the ventricle in the posterior margin of the infundibulum.

When compared to the left ventricle, the right ventricular wall is thin and measures 3-4 mm at end diastole. Further thinning of the free wall towards the RV apex is normal. Enlargement of

the RV chamber results in obliteration of the retrosternal clear space and because this space is a small space, the resultant effect of further enlargement is that there is displacement of the left ventricle leftward and posteriorly, and the apex backward and superiorly.

Left ventricle: Cone shaped thick walled chamber with its long axis in the left anterior and inferior direction. It forms the inferior part of the left heart border, part of the posterior heart border and the apex. Its base is formed by the fibrous skeleton of the inlet and outlet valves and its apex is the apex of the heart. Characteristic features that distinguish the left ventricle are 1) fibrous continuity of the inlet and outlet valves due to absence of conus or infundibulum, 2) two papillary muscles – the anterolateral and the posteromedial papillary muscles that arises from only the free wall of the ventricle, 3) absence of features that are characteristic for right ventricle.

The myocardial thickness in the left ventricle measured in the lateral wall at end diastole is 7-8 mm in women and 8-9 mm in men. The apex is thinner and measures about 3 mm. Wall thickness is not uniform; it is most pronounced in the longitudinal direction with gradual thinning toward the apex and less so in the circumferential direction.

Ventricular septum: The interventricular septum is a thick walled muscular layer that separates the right ventricle from the left ventricle. At the subaortic location the muscular layer is thin and is called the membranous septum. The interventricular septum is convex shaped towards the right ventricle which is maintained throughout the cardiac cycle.

Valves:

The mitral valve is always connected to the morphological left ventricle and the tricuspid valve to the morphological right ventricle. The tricuspid valve is more apically positioned in comparison to the mitral valve; this allows differentiation of the ventricular morphology. The tricuspid valve has 3 leaflets, septal, inferior and anterosuperior leaflets. The mitral valve has 2 leaflets, aortic and mural leaflets. The semilunar valves attach at the anatomic ventriculoatrial junction. Both have three cusps.

The valve leaflets are thin and fibrous therefore the leaflet morphology, motion and abnormal opening and valvular flow patterns may not be optimally assessed.

Pericardium:

The pericardium covers the heart along with the origin of the great vessels. It consists of the fibrous part and the serous part. The fibrous part is attached to the sternum and the diaphragm. The serous part consists of the visceral layer that is in contact with the heart and the pericardial fat and the parietal part which is in contact with the inner surface of the fibrous pericardium. The visceral layer reflects at the heart and the root of the great vessels onto the inner surface of the fibrous part of the pericardium thus becoming continuous with it. The pericardial cavity lies between the two layers of the serous part of the pericardium and contains about 20-25 ml of serous fluid. This may vary in individuals. Two serosal tunnels are present- the transverse sinus, posterior in location to the great arteries and anterior to the

atria and superior vena cava with its four recesses, the superior aortic recess, inferior aortic recess, left pulmonic recess, right pulmonic recess, and the oblique sinus, posterior in location to the left atrium.

On magnetic resonance, the normal pericardium is seen as an extremely thin hypointense curvilinear structure with hyperintense mediastinal and epicardial fat on both sides. Measures 1.2+/- 0.5 mm in diastole and 1.7+/-0.5 mm in systole. Thickening of the pericardium the measures more than 4 mm is significant.(28)

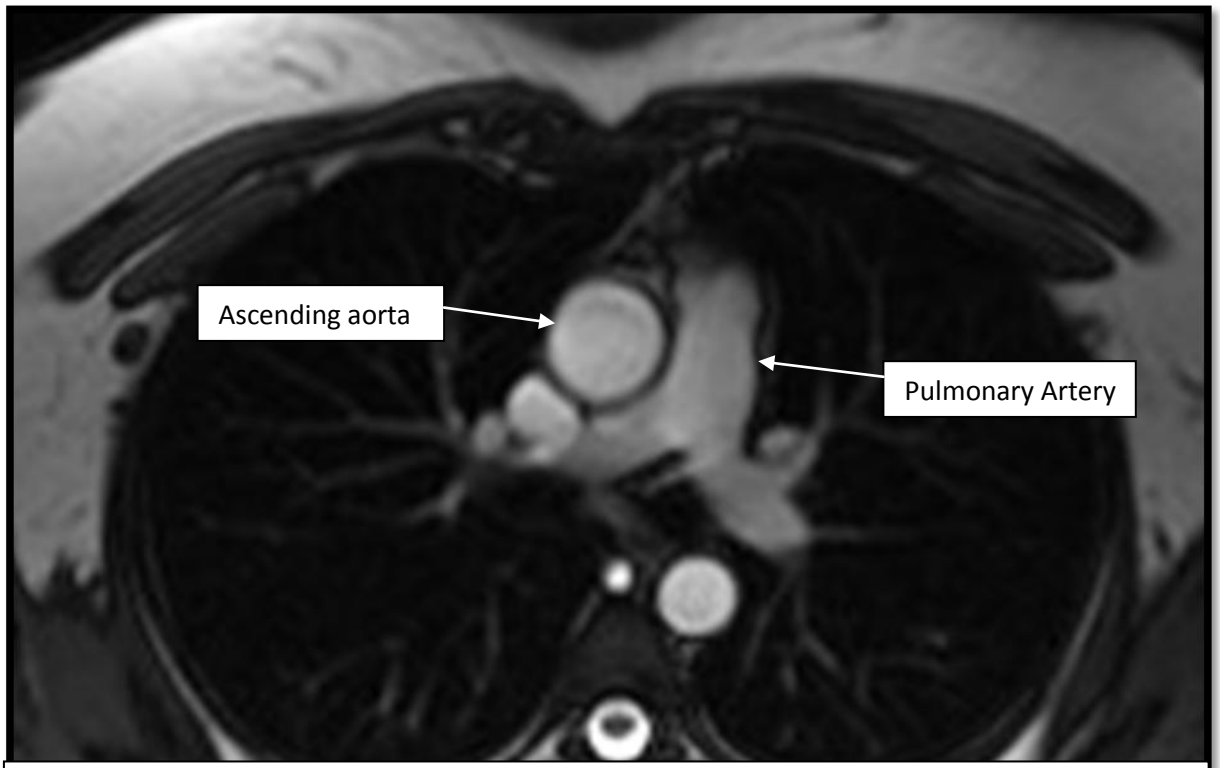


Fig 5.1 MRI TRUFISP axial sections of the chest at the level of the pulmonary artery showing normal anatomy

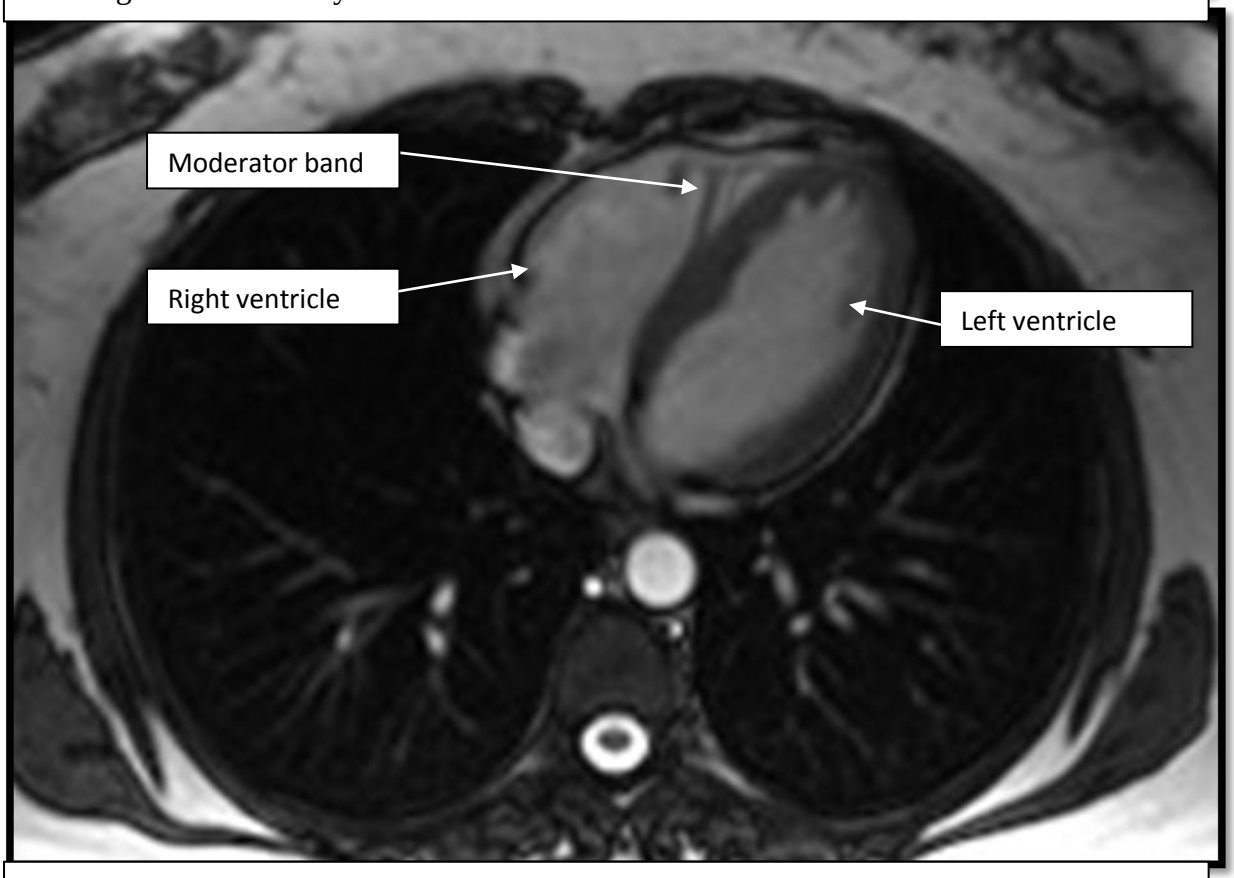


Fig 5.2 MRI TRUFISP axial sections of the chest at the level of heart chambers showing normal anatomy

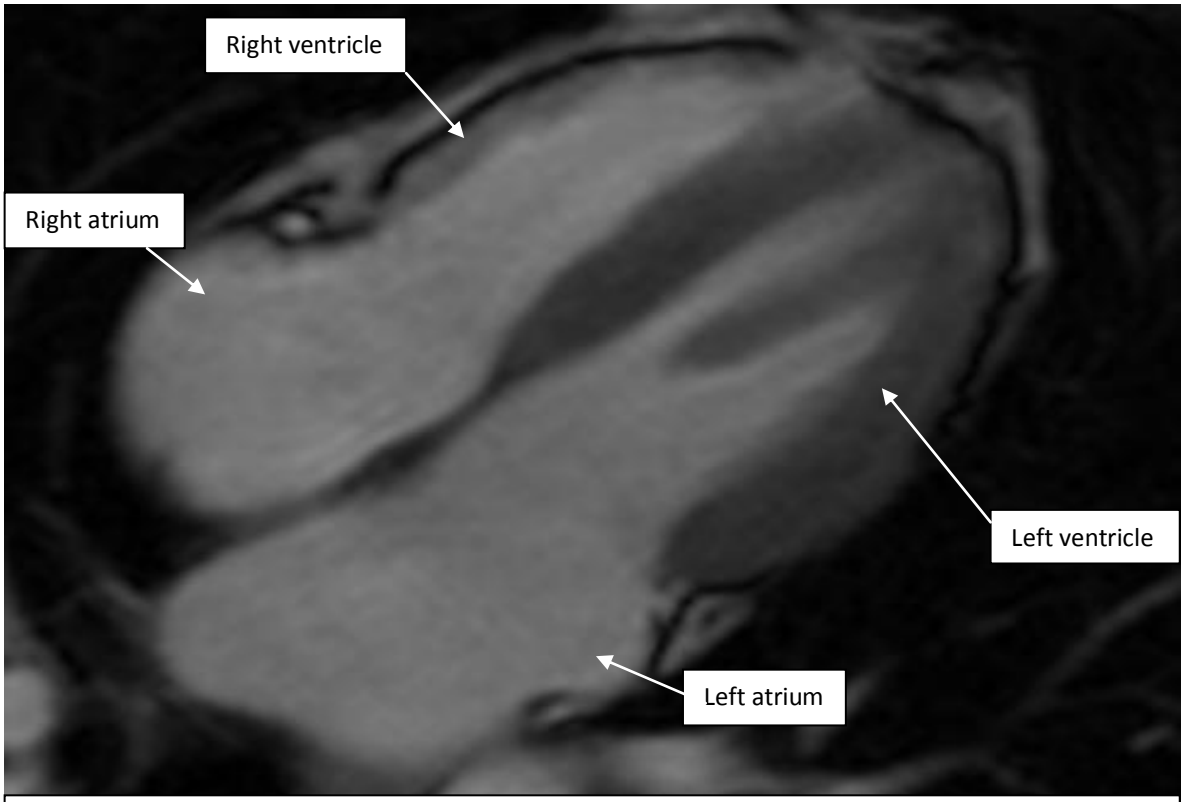


Fig 5.3 MRI CINE four chamber (horizontal long axis) view at systole

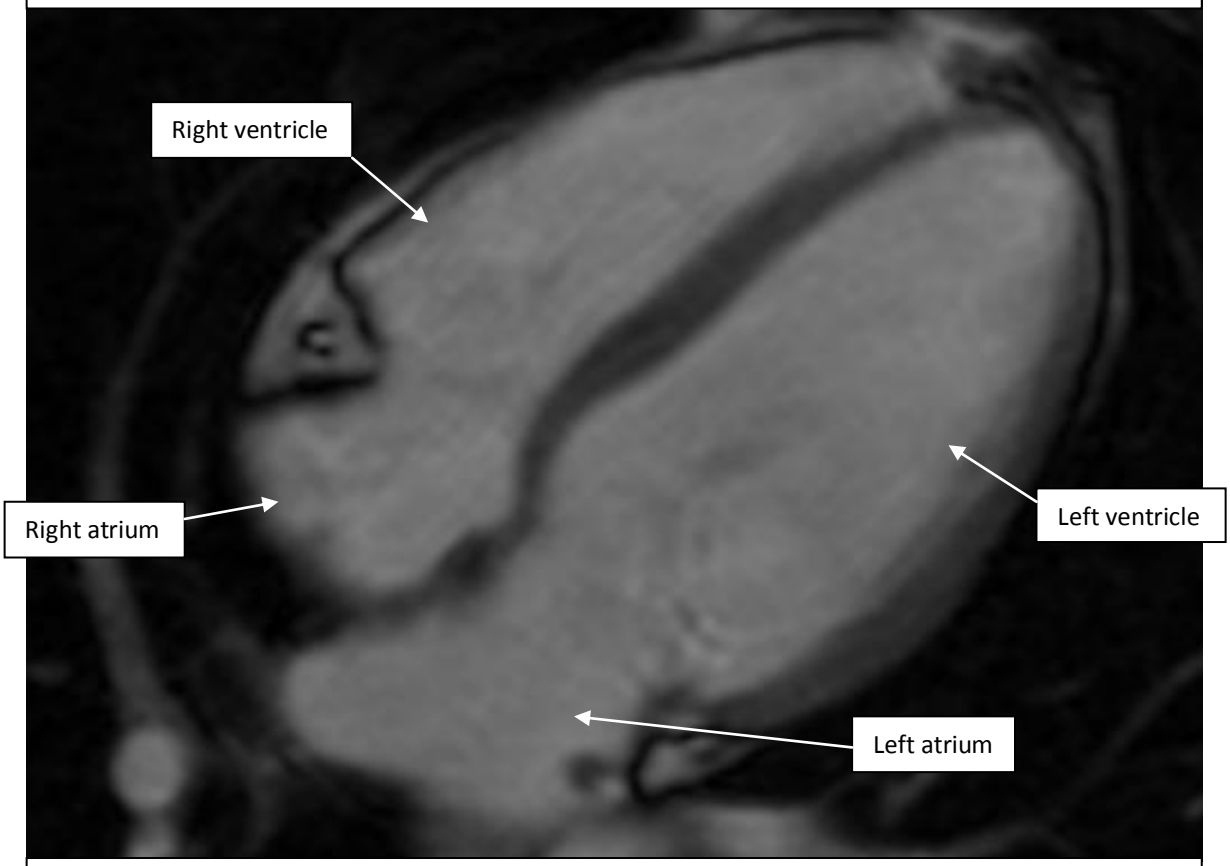


Fig 5.4 MRI CINE four chamber (horizontal long axis)view at diastole

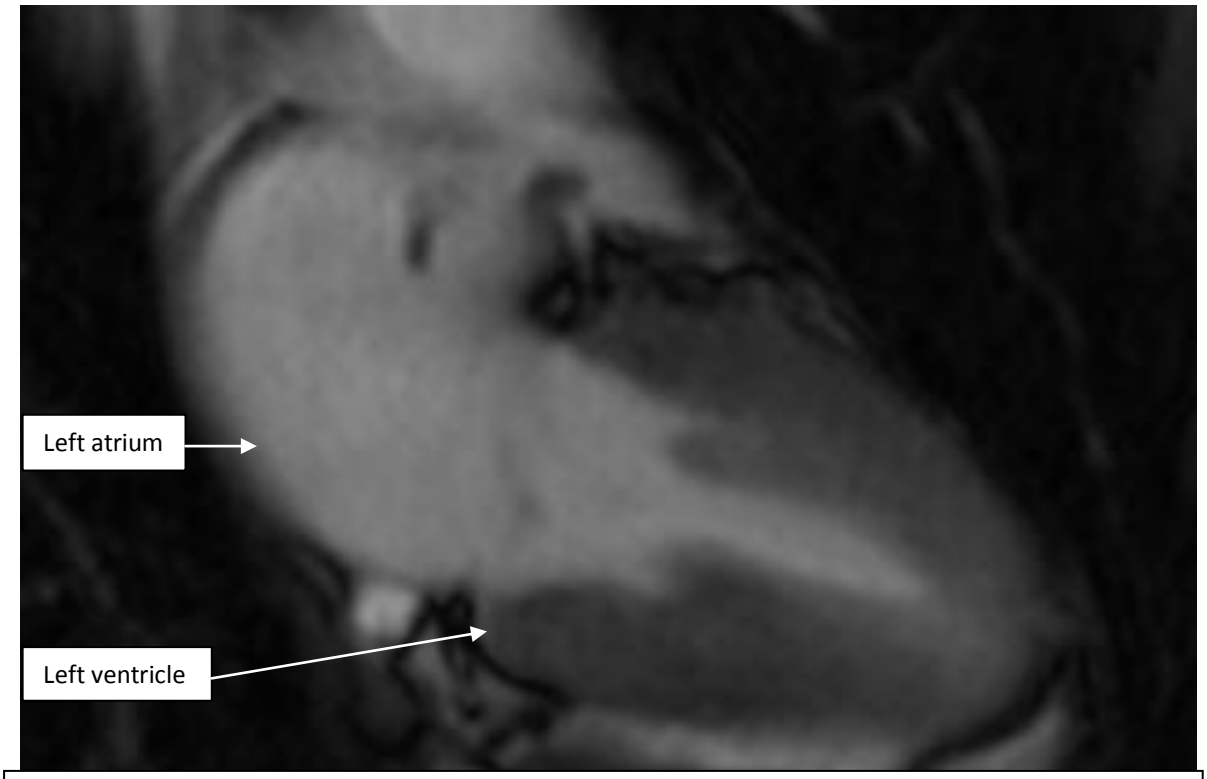


Fig 5.5 MRI CINE two chamber view (vertical long axis view) of left ventricle at systole

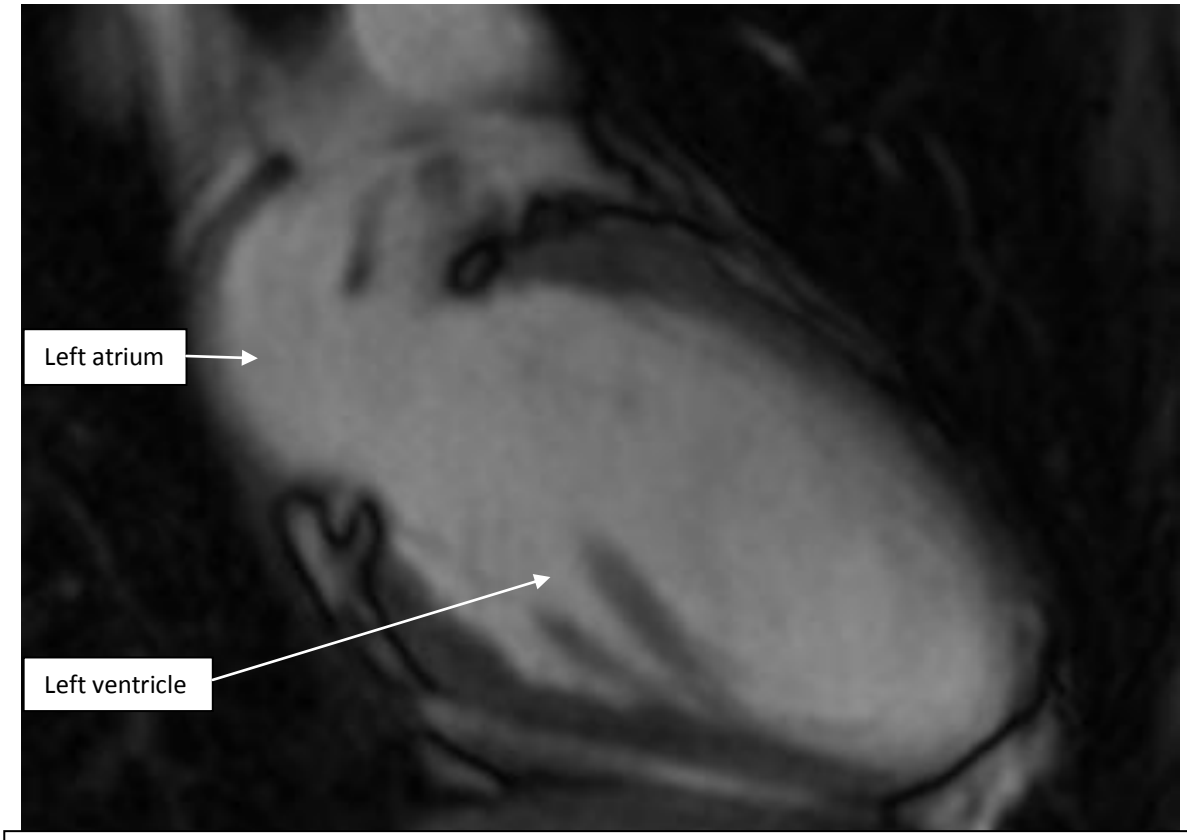


Fig 5.6 MRI CINE two chamber view (vertical long axis view) of left ventricle at diastole

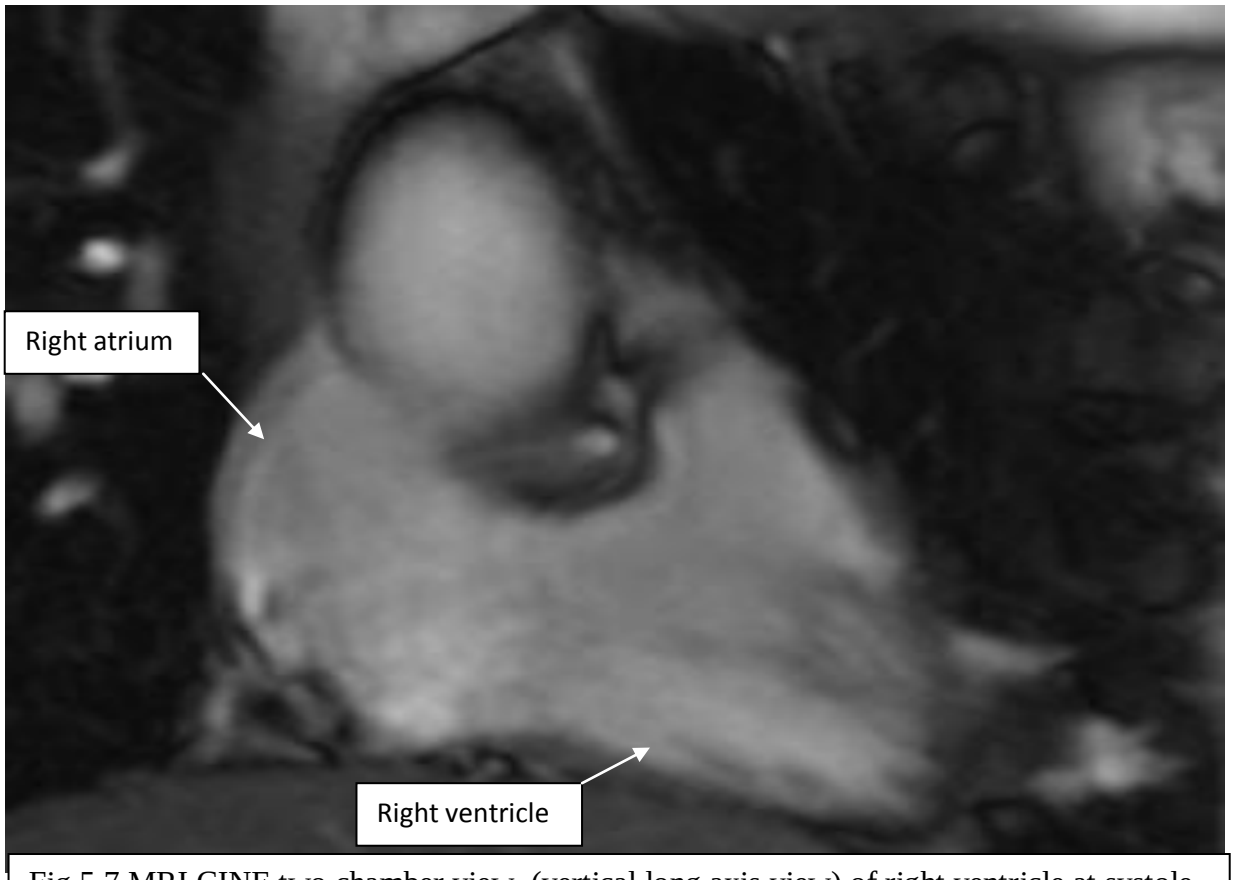


Fig 5.7 MRI CINE two chamber view (vertical long axis view) of right ventricle at systole

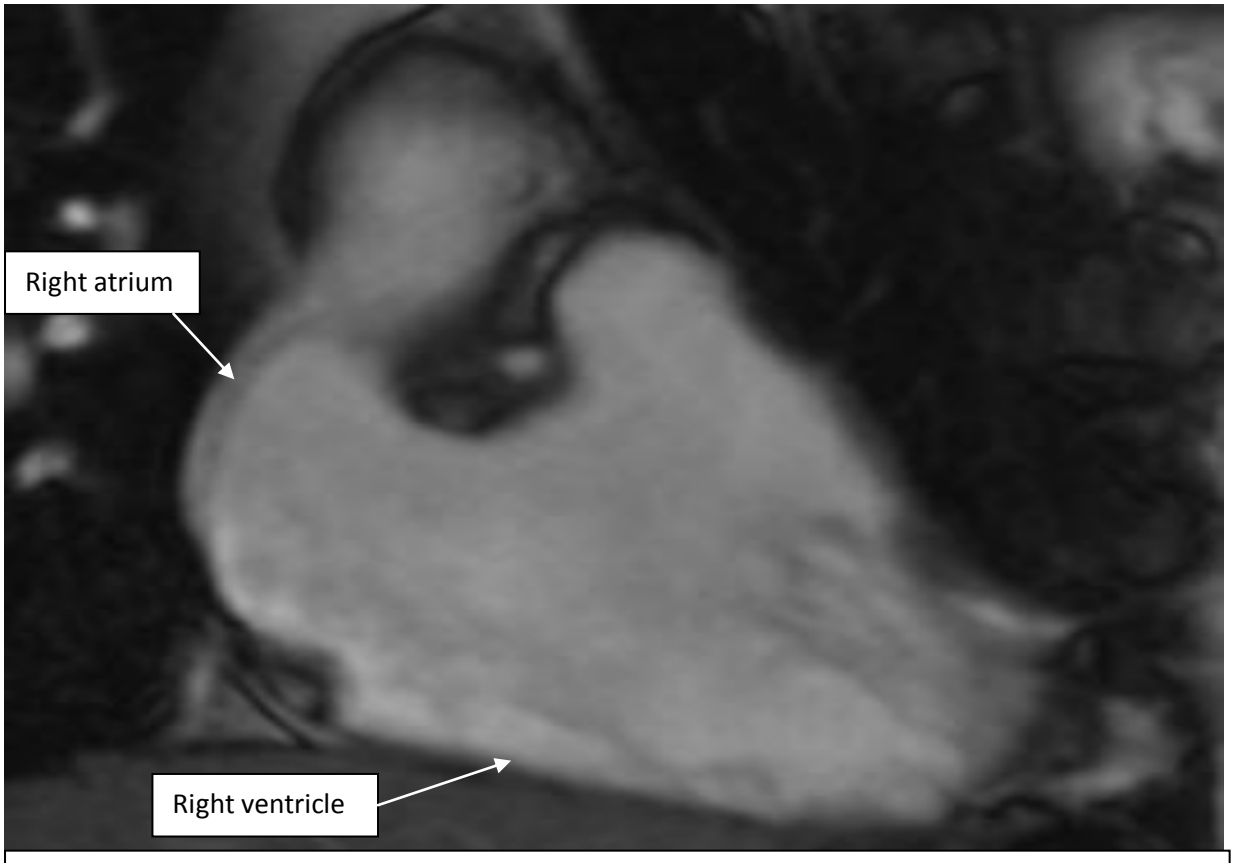


Fig 5.8 MRI CINE two chamber view (vertical long axis view) of right ventricle at diastole

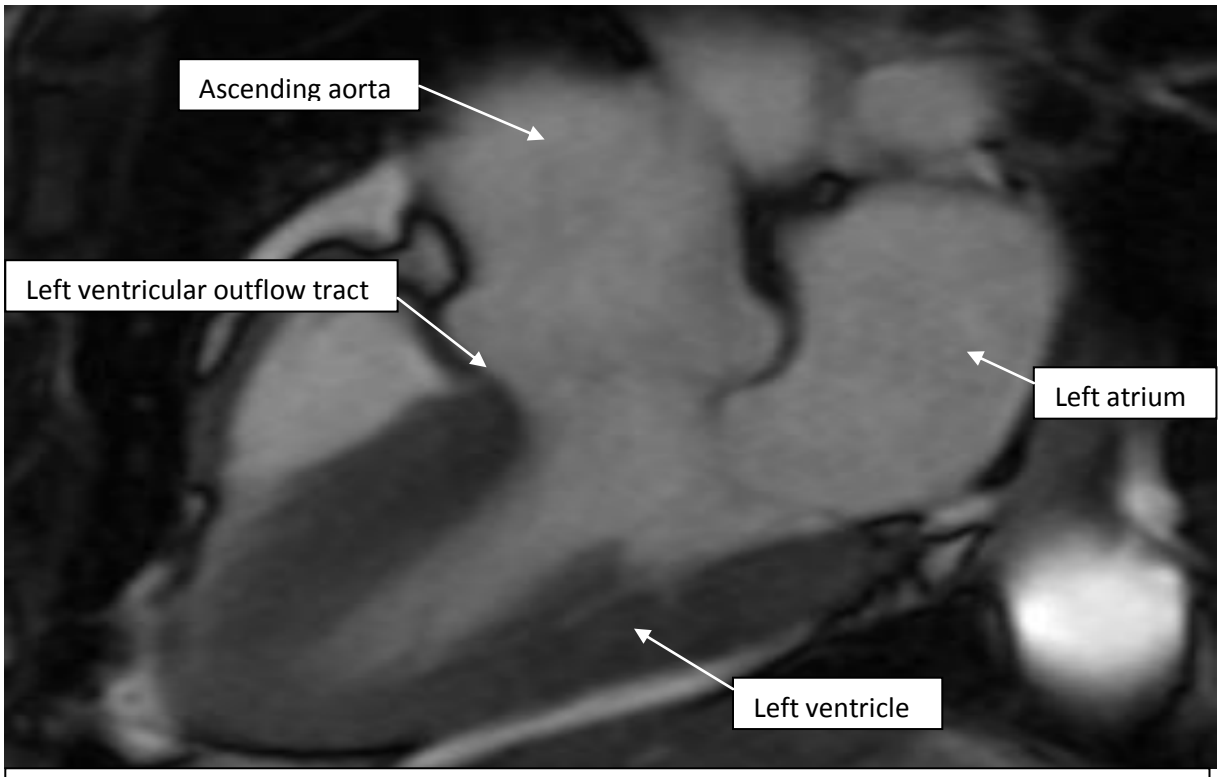


Fig 5.9 MRI CINE left ventricular outflow tract at systole

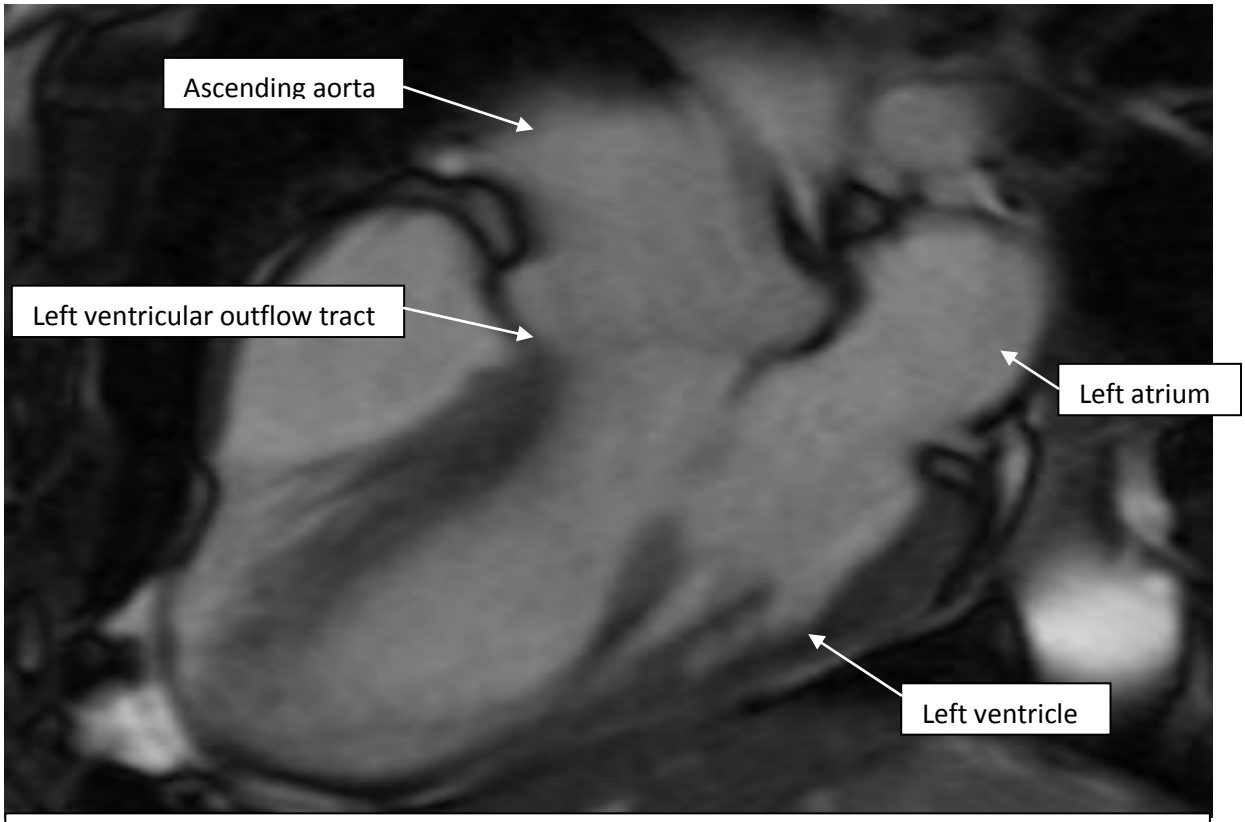
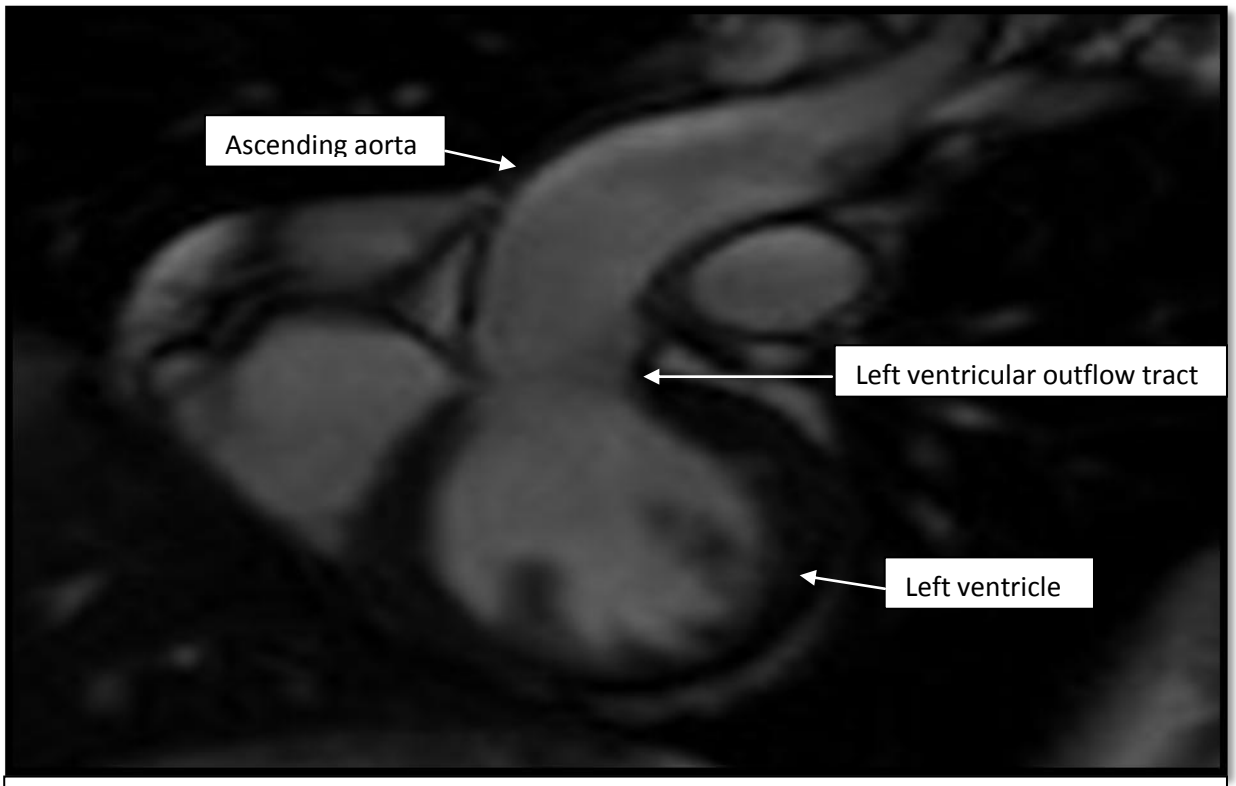
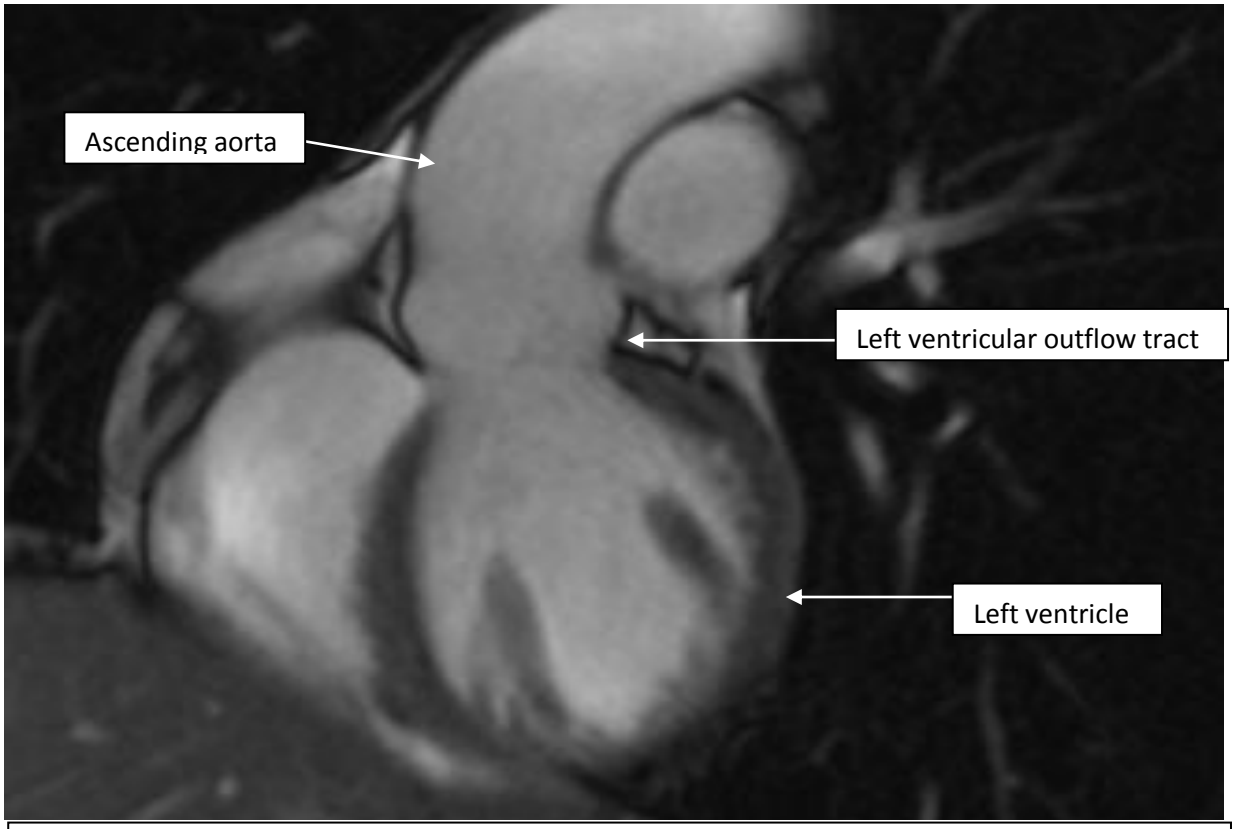


Fig 5.10 MRI CINE left ventricular outflow tract at diastole



5. 11 MRI CINE left ventricular outflow tract at systole



5. 12 MRI CINE left ventricular outflow tract at diastole

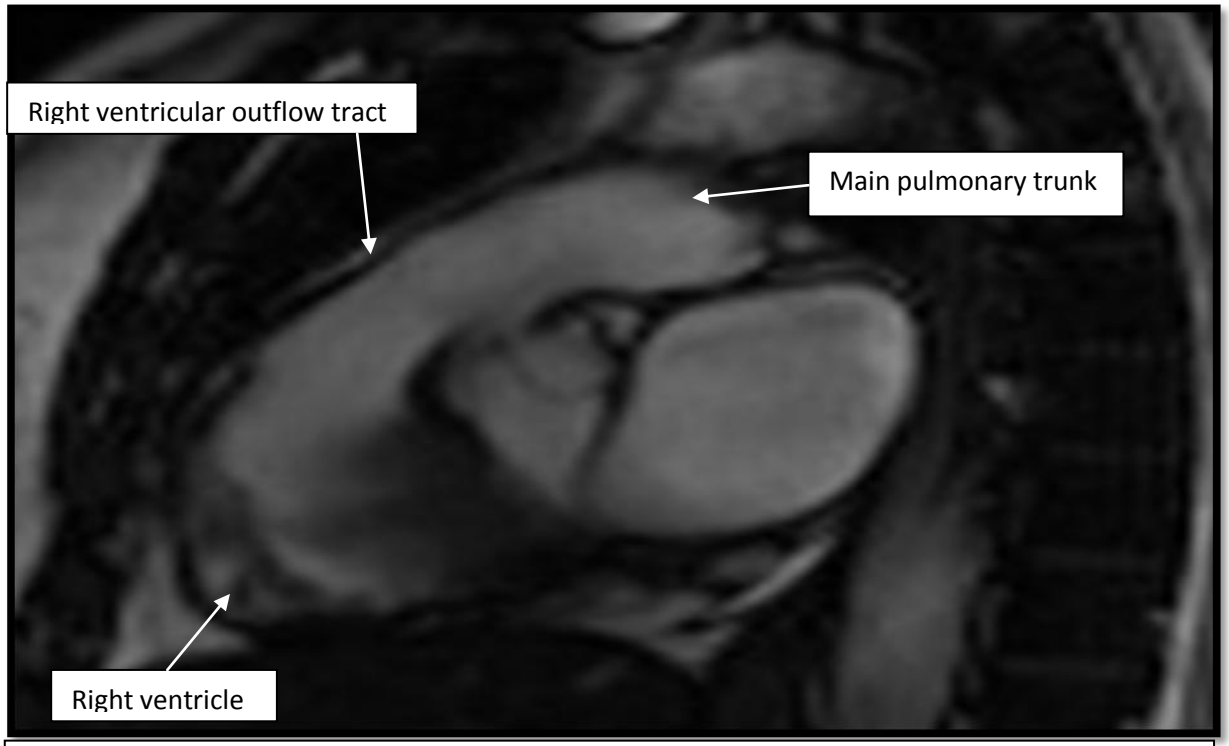


Fig 5.13 MRI CINE right ventricular outflow tract at systole

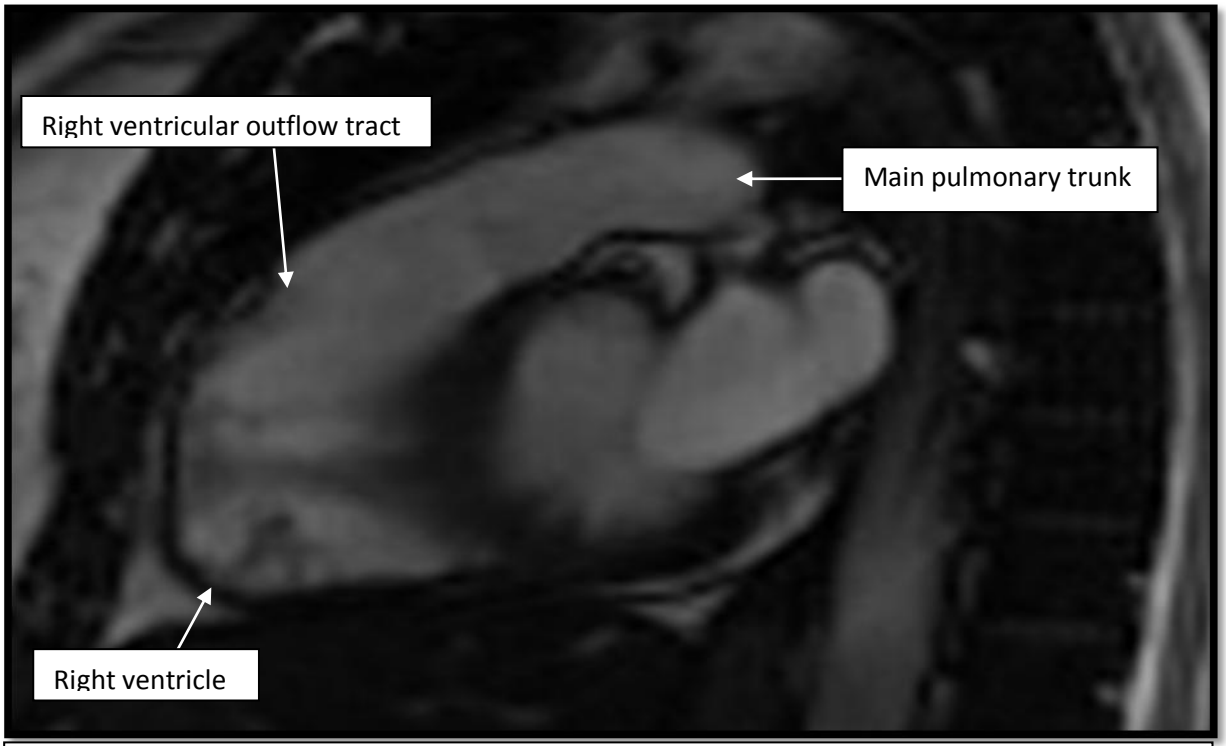


Fig 5.14 MRI CINE right ventricular outflow tract at diastole

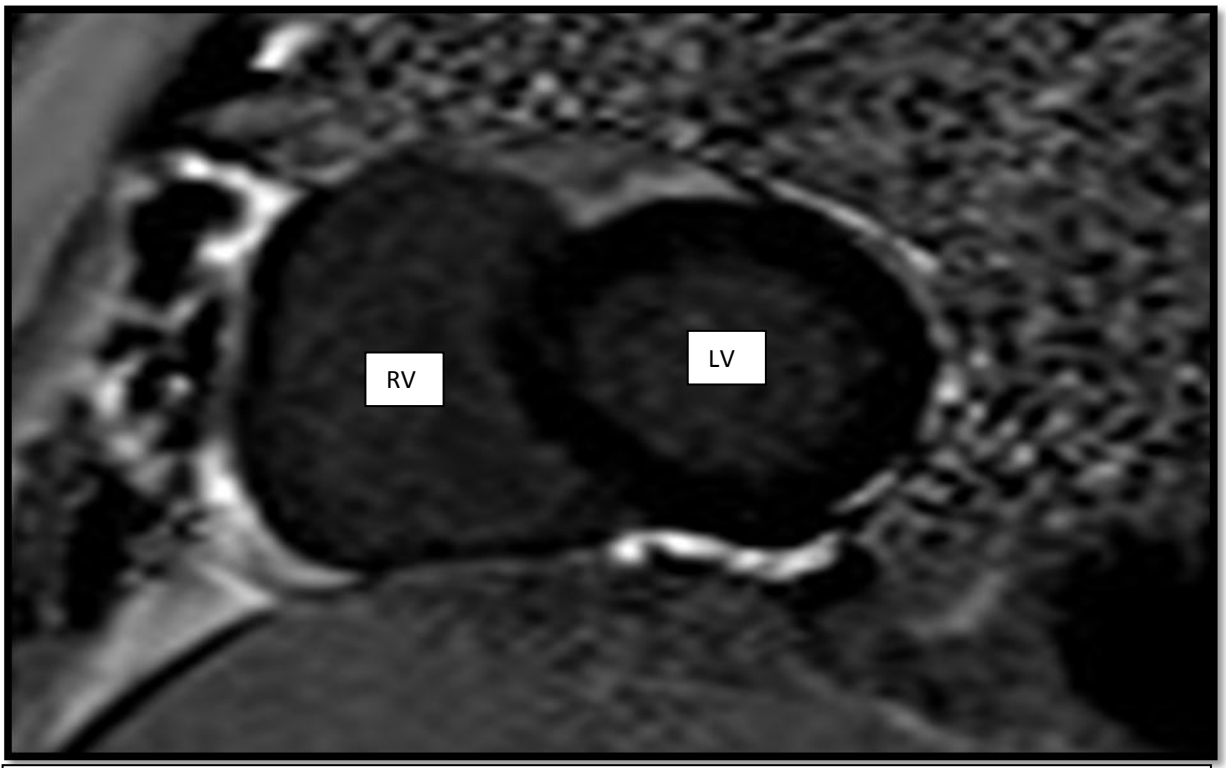


Fig 5.15 PSIR sequence short axis at basal level

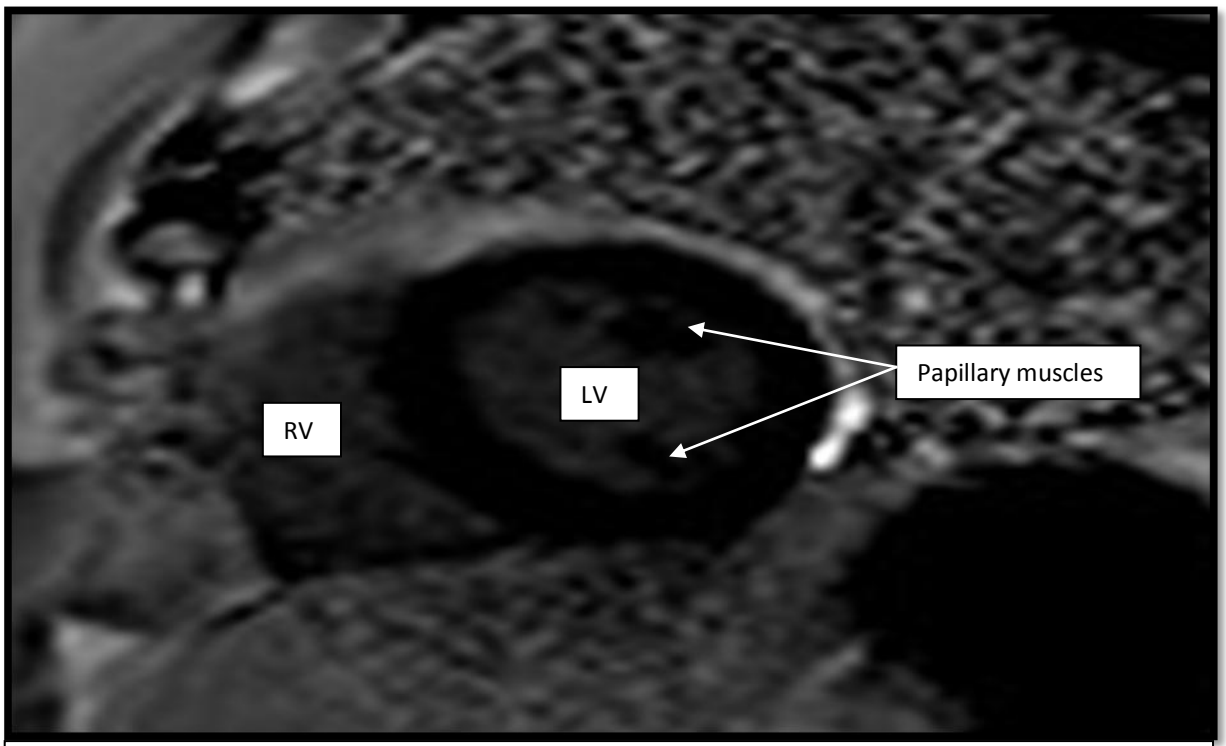


Fig 5.16 PSIR sequence short axis at midcavity level

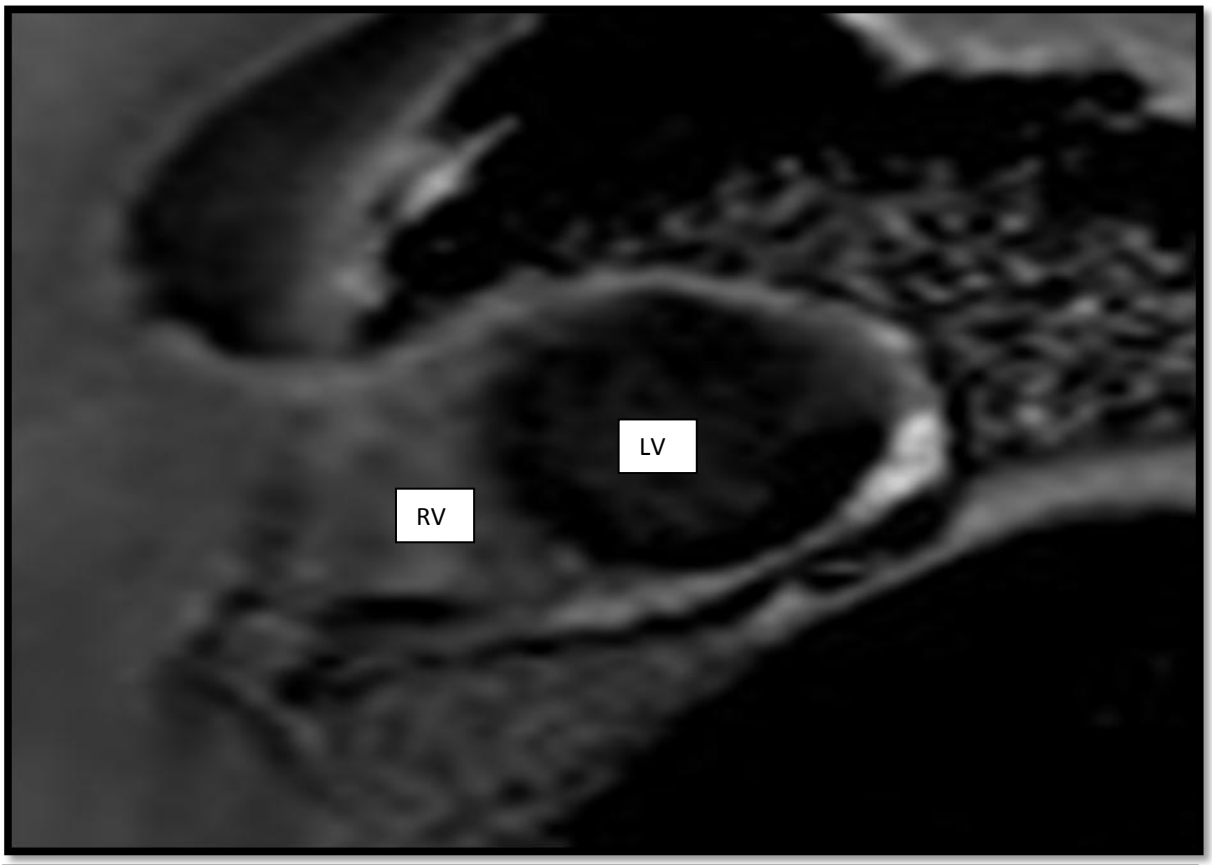


Fig 5.17 PSIR sequence short axis at apical level

Fig 5.18 PSIR sequence four chamber

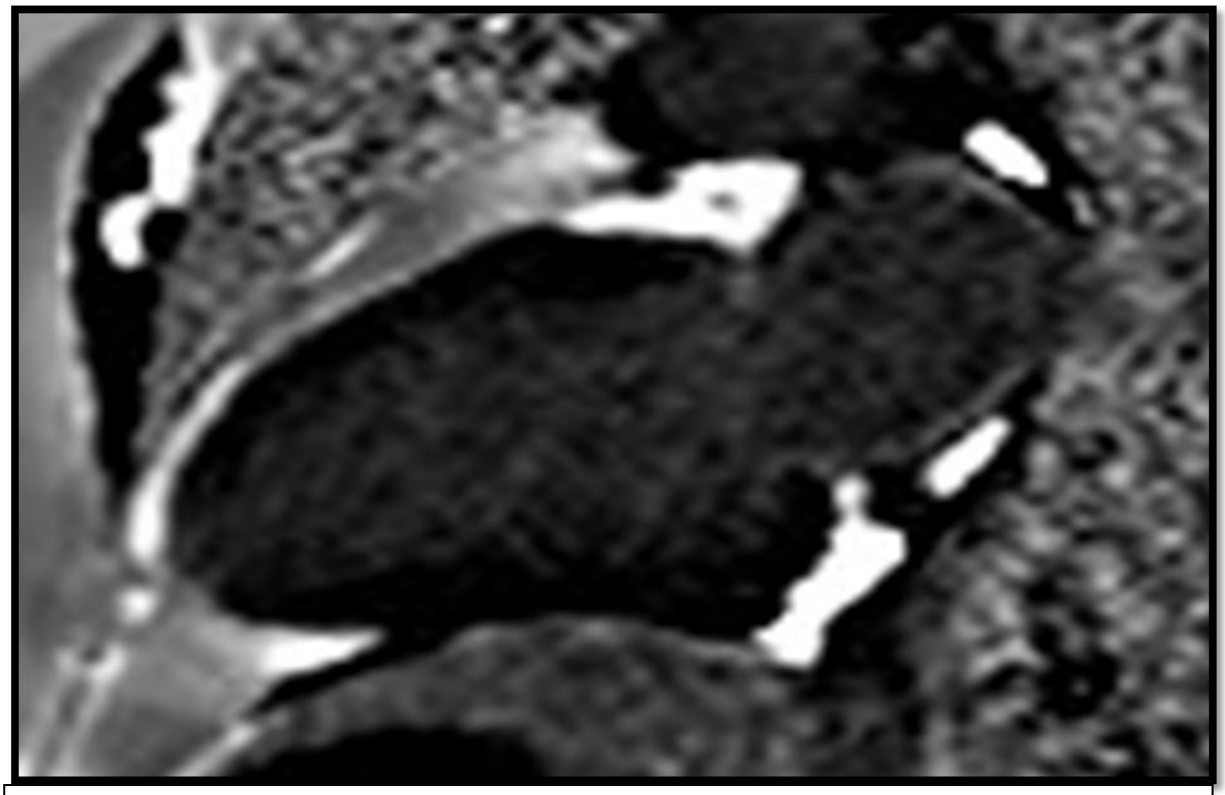


Fig 5.19 PSIR sequence two chamber left ventricle and left atrium

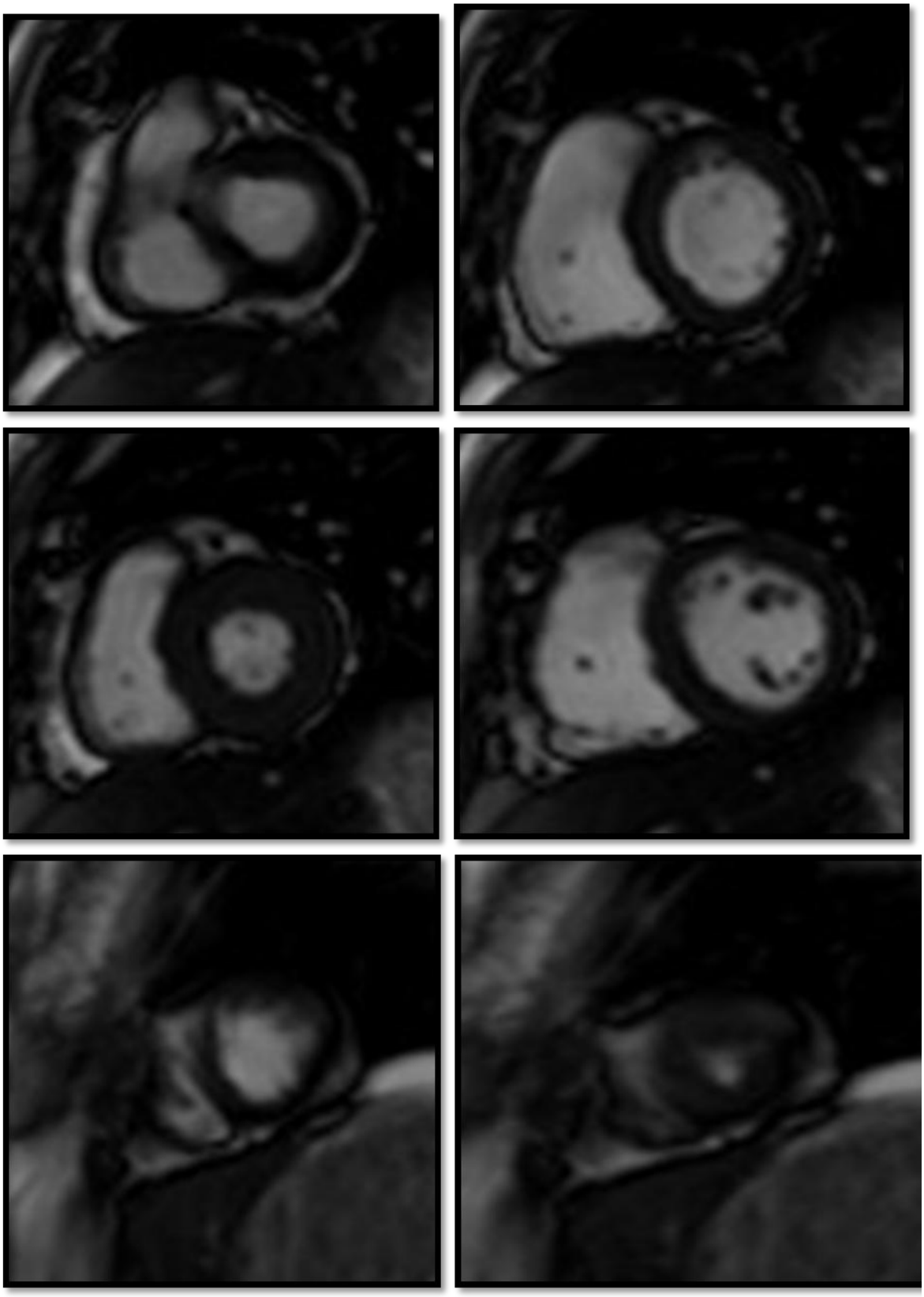


Fig 5.20 MRI CINE short axis at systole and diastole- basal, midcavity and apical level

Coronary arteries: The heart is supplied by coronary arteries that have a characteristic high capillary density distal vascular territory. The amount of blood passing through coronary arteries is about 250 ml/min or about 1ml/ min/ gram. This constitutes 5% of cardiac output. Coronary flow is greater during diastole than during systole unlike elsewhere in other parts of the body. During maxima exercise, the myocardial blood supply increases by 4 to 6 times, obtained by dilatation of the coronary arteries.(4)

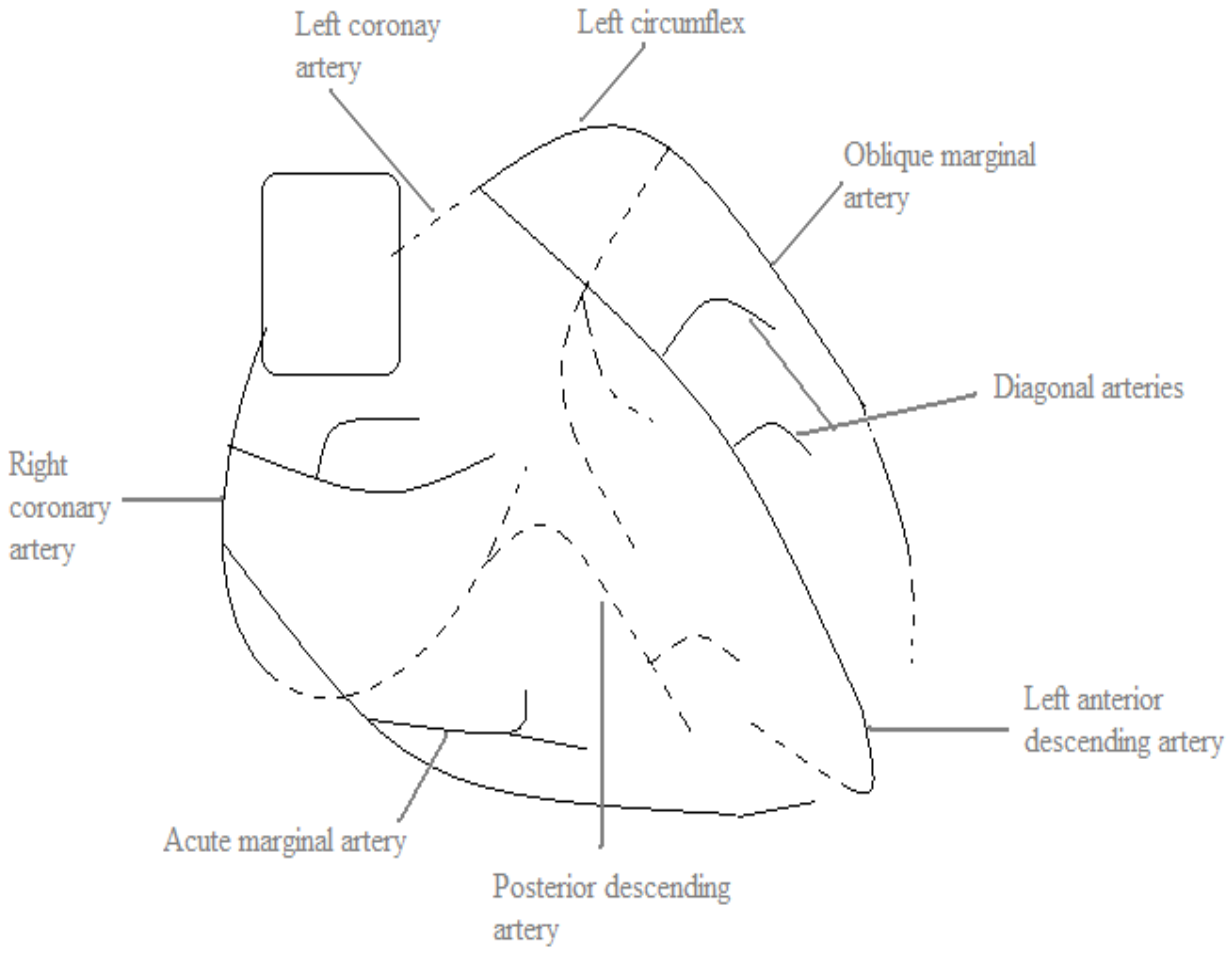


Fig 6 Schematic diagram of coronary arteries

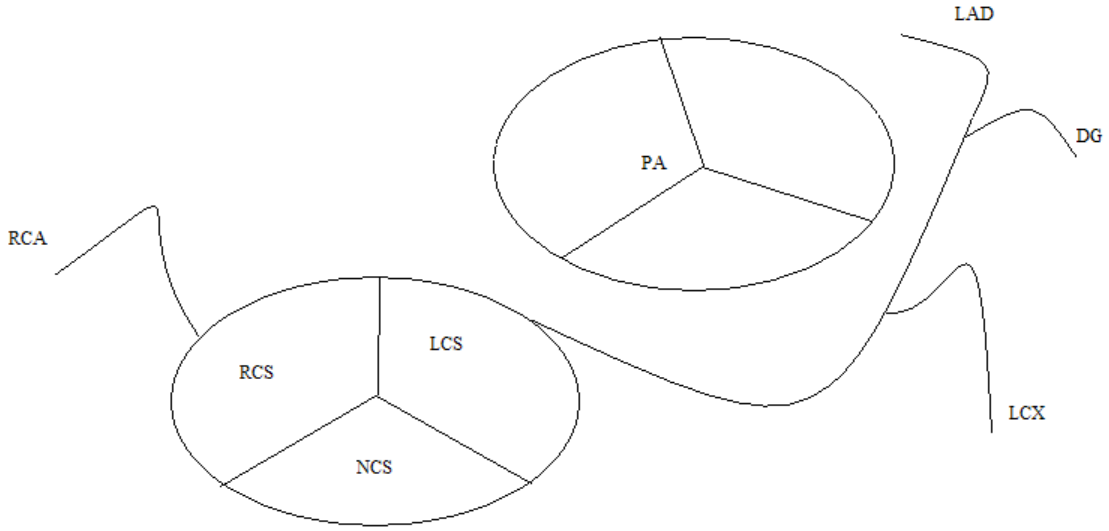


Fig 6.1 Schematic diagram of coronary arteries

There are two major arteries the left main coronary artery (LMCA) and the right coronary artery (RCA).

The left coronary artery originates in the left sinus of Valsalva and its length is variable. After a length of 10 to 15 mm it bifurcates into the left anterior descending artery (LAD) and circumflex artery (LCx). It may trifurcate, the third branch being the intermediate ramus branch. In 0.41% of the population, the left main coronary artery is absent.

The left anterior descending artery is located in the anterior interventricular groove and gives rise to several septal branches and diagnonal branches. Septal branches supply the anterior two third of the interventricular septum and the diagonal branches supply the lateral aspect of the left ventricle.

The left circumflex artery is located in the left atrioventricular groove and supplies the lateral and posterolateral aspect of the left ventricle. 10 to 20% of the population have left dominant circulation in which case the left circumflex artery supplies the posterior descending coronary artery and the posterior left ventricular artery.

The right coronary artery originates in the right coronary sinus of Valsalva. It is located in the right atrioventricular groove. The first branch is the conus branch. The acute marginal branches of the right coronary artery supplies the lateral aspect of the right ventricle. 70 to 80 % of the population have a right dominant circulation in which case, in the atrioventricular groove the right coronary artery supplies the posterior descending artery, the posterior left ventricular artery and sometimes a third branchwhich is the posterolateral artery.

In the 10 to 20 % of the population having a left dominant circulation one of the following 3 is adopted by the right coronary artery. 1) Coronary segment 1 has a normal diameter and coronary segments 2 and 3 are hypoplastic. 2) Coronary segments 1,2 and 3 are hypoplastic. 3) Coronary segments 1, 2, and 3 have a small uniform diameter.

Coronary anomalies: Minor coronary anomalies are common and are clinically not significant. The most common is the left circumflex artery arising from the right aortic sinus. This can occur in upto 50% of the population. The left circumflex coronary artery can arise from the right sinus of Valsalva independently or in a common ostium with the right coronary artery. It may also arise directly from the right coronary artery. Another common anomaly is the absence of the left main coronary artery and independent origin or sharing of a common

ostium between the left anterior descending artery and the left circumflex artery from the left sinus of Valsalva.

Most of the major coronary anomalies have ectopic origin in the contralateral side of the heart. They lie between aorta root and the pulmonary trunk. This location predisposes the lumen of the origin and proximal aspect of the artery thereby resulting in significant hemodynamic changes that can even result in sudden cardiac death. The anomalies can be as follows. 1) RCA arises from the left sinus of Valsalva and courses between the aortal and pulmonary trunk. 2) The left main coronary artery arises from the right sinus of Valsalva and courses between the aorta and the pulmonary trunk. 3) Origin of the left main coronary artery, left anterior descending or right coronary artery from the pulmonary trunk. 4) Coronary artery fistula with communication between the coronary arteries and a cardiac chamber or a systemic vein.

Myocardial bridging is a congenital anomaly of the coronary arteries in which a segment of a major epicardial coronary artery runs intramurally through the myocardium. It is also known as the tunneled artery. In a small percentage of the population having this anomaly, it may cause angina symptoms or myocardial ischemia.(28)(29)

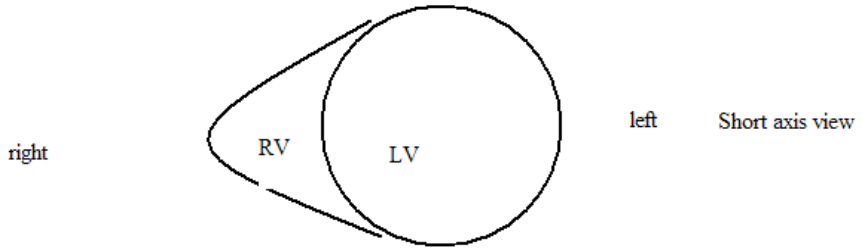
Planes for MR imaging of the heart

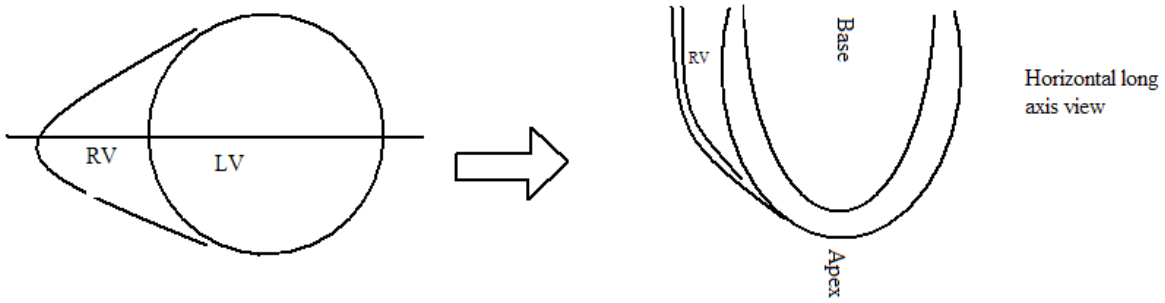
Scout images: Scout image is taken in the axial plane. Axial images display normal anatomy and relationship of the great vessels and cardiac chambers and the pericardium depicting the morphology in a plane that is familiar to general radiologist. However quantitative

measurement of wall thickness, cavity dimensions and functional data cannot be assessed. Images can also be taken in any desired orthogonal views.

The heart lies oblique in the thoracic cavity. The true long axis of the hear is oriented 45 degrees to the midsagittal plane of the thoracic spine. To obtain correct inclinations for imaging along the cardiac axes, the following is done:

- vertical long axis plane: on an axial scout, a plane is chosen that runs through the apex of the left ventricle an dthe middle of the left atrioventricular valve which is the mitral valve.





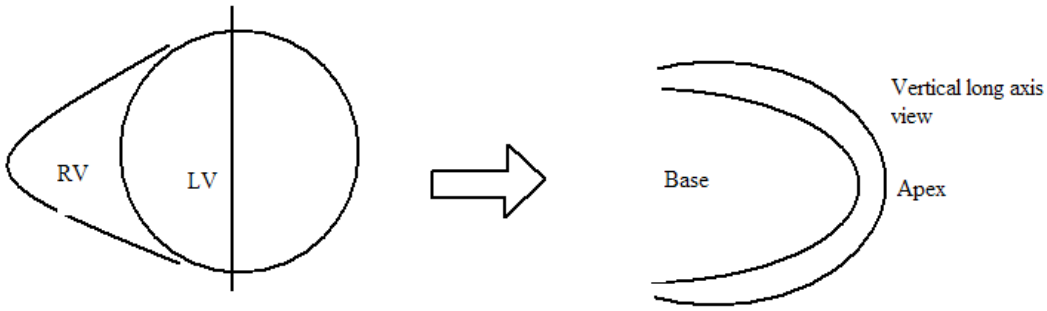


Fig 7 – Schematic diagram showing different planes in imaging of the heart and the nomenclature of the sections

- horizontal long axis / four chamber view: on the vertical long axis, a plane is chosen that runs through the apex and the middle of the mitral ring.

- short axis plane: the short axis plane is taken as that which is perpendicular to the horizontal long axis and vertical long axis plane.

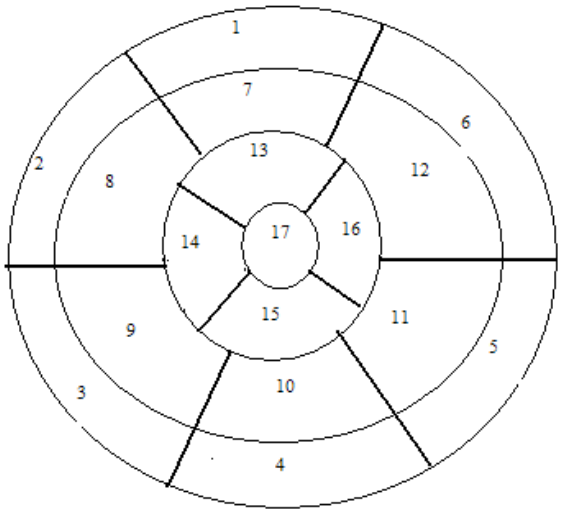
- true 4 chamber view: in a short axis view, a plane is taken that passes from the superior most mitral valve, anterolateral papillary muscle to the inferior angle of the right ventricle anteriorly and through the mid point of the interventricular septum, to get the true 4 chamber view.

- true short axis plane: is taken perpendicular to the true 4 chamber view. No single plane is absolutely perpendicular to both the ventricular walls however the plane that is positioned parallel to the mitral valve between the anterior and posterior atrioventricular groove is best.

- from the true short axis plane, the vertical long axis, horizontal long axis, the left ventricular inflow / outflow can be acquired.

Segmentation of the left ventricle

The left ventricle can be divided into 17 segments so as to allow accurate description of anatomy and abnormality in a standardized manner between various cardiovascular imaging modalities, as follows:



1. basal anterior
2. basal anteroseptal
3. basal inferoseptal
4. basal inferior
5. basal inferolateral
6. basal anterolateral
7. mid anterior
8. mid anteroseptal
9. mid inferoseptal
10. mid inferior
11. mid inferolateral
12. mid anterolateral
13. apical anterior
14. apical septal
15. apical inferior
16. apical lateral
17. apex

Fig 8 Schematic diagram of segmentation of the LV

The heart is divided into three sections of equal longitudinal length as shown, comprising of the basal level, the midcavity level and the apical level. The basal and midcavity level each is divided into six equal segments each line dividing the segments drawn at 60 degrees angle to one another. The apical level is divided into four equal segments each line dividing the segments drawn at 80 degrees angle to one another. The papillary muscles are anatomical land marks to differentiate between the mid cavity level from the basal and apical levels. The apex is the 17^{th} segment which is defined by the absence of cavity, that is, the apex is the muscular tip of the left ventricle. The apex is visualized on the long axis images.

The segments can be used to relate the most common distribution of coronary artery anatomy to correlate regional wall motion, perfusion, and / or late enhancement abnormalities to specific coronary artery territories.

Cardiac function:

Cine MRI is used to visualize cardiac motion. It is similar to echocardiography. The main advantage of cine MRI in the myocardial wall motion evaluation and LV function when compared to echocardiography is its ability to sharply delineate endocardial borders. This superior quality of cardiac MRI allows us to interpret and quantify more accurately and consistently the LV function. Of course the question of poor acoustic window does not arise with cardiac MRI. In one particular study that assessed quality of the images taken in 208 patients as part of its analysis, showed that only 51% of the echocardiograms were considered to be of good or very good image quality, whereas 82% of the MR equivalents were graded highly.(30)

Modified Simpson rule- volume of an object can be estimated by taking the sum of the cross sectional areas of each section and multiplying by the section thickness.

A stack of short axis cine images is taken and the volume is assessed at end systole and end diastole. Ejection fraction is calculated as the difference of LV volumes at end systole and end diastole divided by the left ventricular volume at end diastole. Papillary muscles are included in the blood pool by consensus. The stacks of short axis images are acquired in the steady state free precision sequence which results in an excellent contrast between the blood pool and the myocardium. This contrast is maintained however ischemic or distorted by disease the chamber is. Therefore there is no requirement for geometric assumption at any point in time, making the assessment of ejection fraction very accurate and the inter study variation very less.

Left ventricular wall motion: The systolic ventricular wall motion of the left ventricle can be evaluated by assessing the contraction of the wall both globally and regionally. Each segment is visualized for several diastolic-systolic motion and the contraction motion of the segment characterized and classified according to the severity of wall motion abnormality. Another method of assessment is by the formula end systolic wall thickness minus end diastolic wall thickness divided by end diastolic wall thickness, the whole total value multiplied by 100. A scoring system classifies according to severity of the wall motion abnormality. Lowest values represent most severe wall motion abnormality. The details of the scoring is elaborated in the methodology section.

Myocardial perfusion:

In myocardial perfusion images, the relative perfusion of the myocardium is assessed by looking at the first transit of a bolus of contrast agent. 3- 5 slices can be imaged per heart beat. This allows whole heart first pass perfusion imaging to be assessed. With rapid GRE sequences, we can qualitatively assess regional blood flow. Regions that fail to enhance in the first pass suggest being supplied by critically stenosed coronary vessels. These regions are said to have perfusion defects. With the help of adenosine one can also assess perfusion reserve of a particular region. Perfusion reserve may be helpful in detection of significant stenosis.(10)

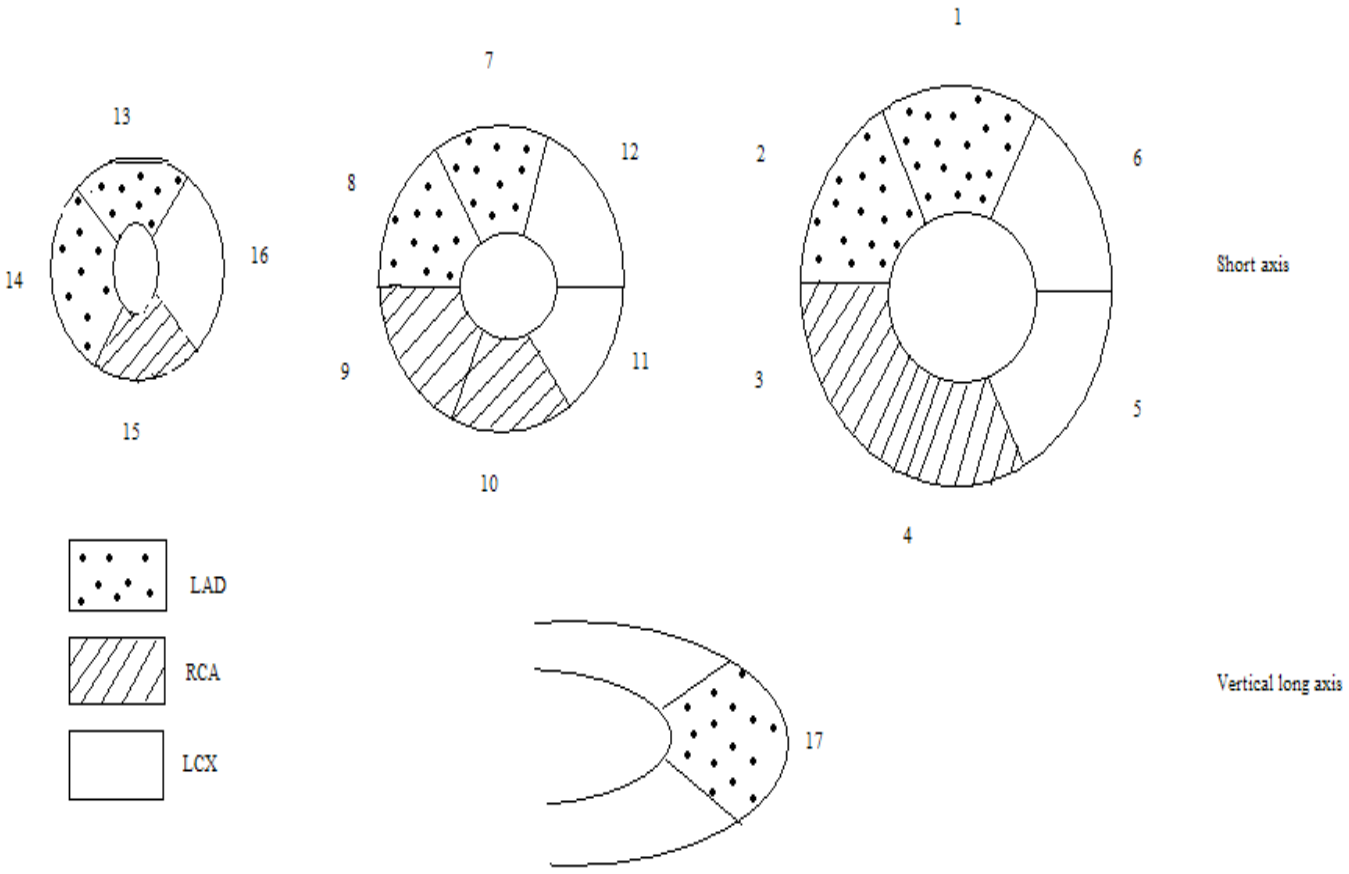


Fig 9 - Assignment of the 17 myocarcial segments to the territories of the left anterior descending (LAD), right coronary artery (RCA) and the left circumflex coronary artery (LCX)

MATERIALS AND METHODOLOGY

STUDY DESIGN: Comparison of semiquantitative method and cardiac MRI in evaluation of myocardial infarct in a patient with ischemic heart disease.

STUDY TYPE: Analytical and descriptive

SETTING: The study was conducted in the Christian Medical College (CMC) Hospital Vellore which is a 2700 bedded tertiary care centre in northern Tamil Nadu. It was established in 1900.

STUDY PERIOD: The study was conducted in the department of Radiology and Cardiology in the period between March 2013 and August 2014.

RECRUITMENT:

INCLUSION CRITERIA: Consecutive patients with a clinical diagnosis of ischemic cardiomyopathy who have undergone an electrocardiography, echocardiography and coronary angiography to diagnose and evaluate the disease were recruited in the study. Patients that need to undergo revascularization are identified by a semiquantitative assessment from a combination of

- clinical data (NYHA grade I, II, III)
- ECG and ECHO showing a myocardial infarct and wall motion abnormality respectively
- subsequent CAG showing arterial lesions >70% lumen occlusion

Patients are thus identified as suffering from coronary artery disease or ischemic heart disease with left ventricle dysfunction.

The patients fulfilling the above inclusion criteria were then informed about the cardiac MRI viability scan and the project in detail.

INFORMED CONSENT: Informed consent was taken by the principal investigator. (Appendix 2)

DATA COLLECTION (Appendix 3):

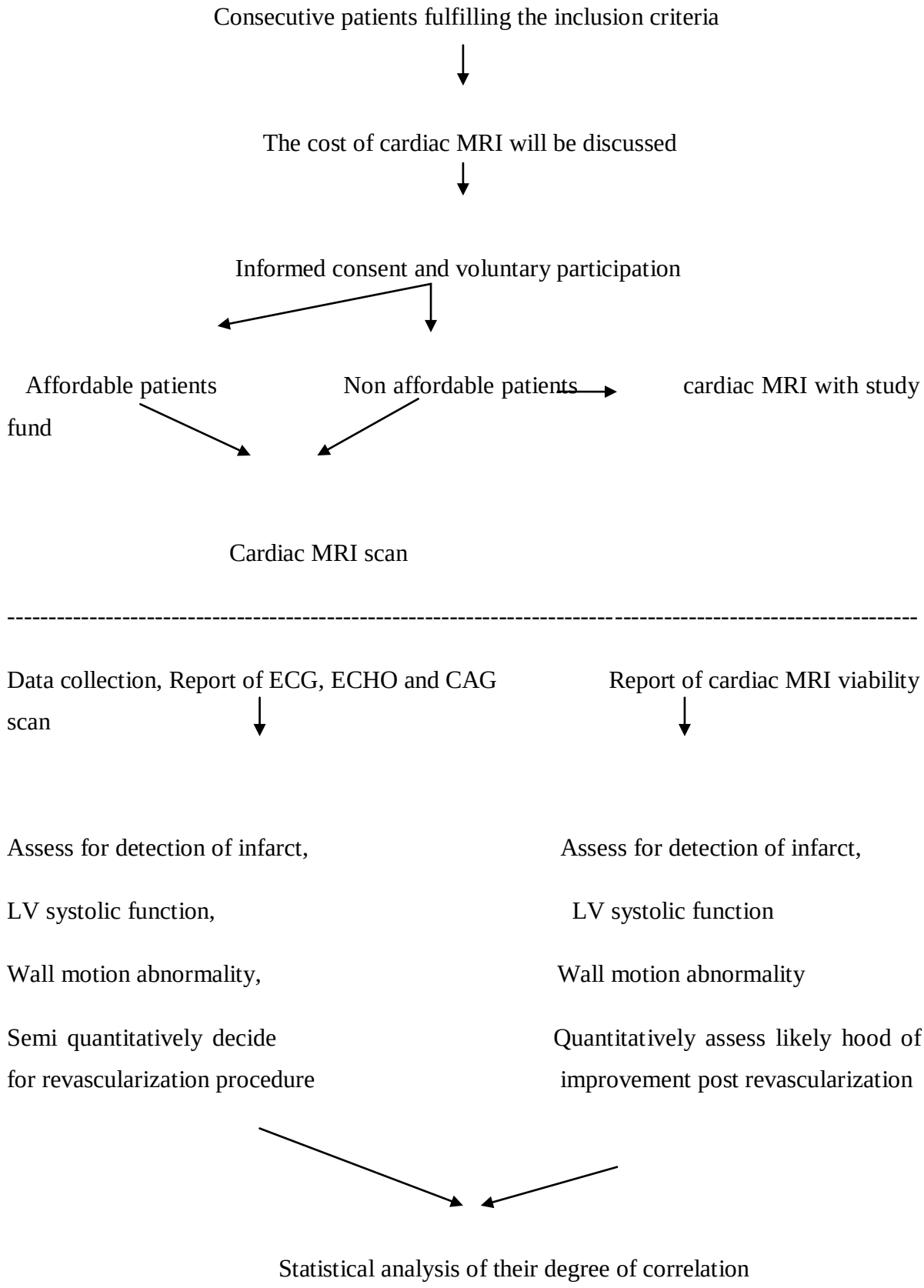
1. Demographic details of the patient was collected.

   Relevant data like history of risk factors (age, sex, family history, smoking, hypertension, dyslipedemia, diabetes mellitus, obesity, physical inactivity, high stress), number of years of disease (according to symptoms), NYHA class and history of treatment (drug therapy / intervention) were recorded.

2. Reporting of electrocardiography, echocardiography and coronary angiogram was done by the co – investigator cardiologist of the concerned unit and the information filled into a standardized format (Appendix 4, 5 and 6). The 17 cardiac segments described by the American Heart Association is used where relevant.

3. The cardiac MRI viability scan was performed in the MRI Room 3, Radiology department with a Siemens 1.5 T MRI machine and the scan was reported by the principal investigator in a standardized format (Appendix 7) and checked by a radiologist of professor grade.

SUMMARY OF THE STUDY METHODOLOGY



PERFORMING THE CARDIAC MAGNETIC RESONANCE IMAGING

IMAGE ACQUISITION:

a) MRI scanner

Cardiac MRI for viability was performed at our institution in a 1.5T scanner (SEIMENS 1.5T Avanto TIM). MRI of the chest in axial section and of the heart in the axis of the heart was done. Cine images were acquired in different views and PSIR phase sensitive inversion recovery sequence was acquired 10 minutes post intravascular contrast injection.

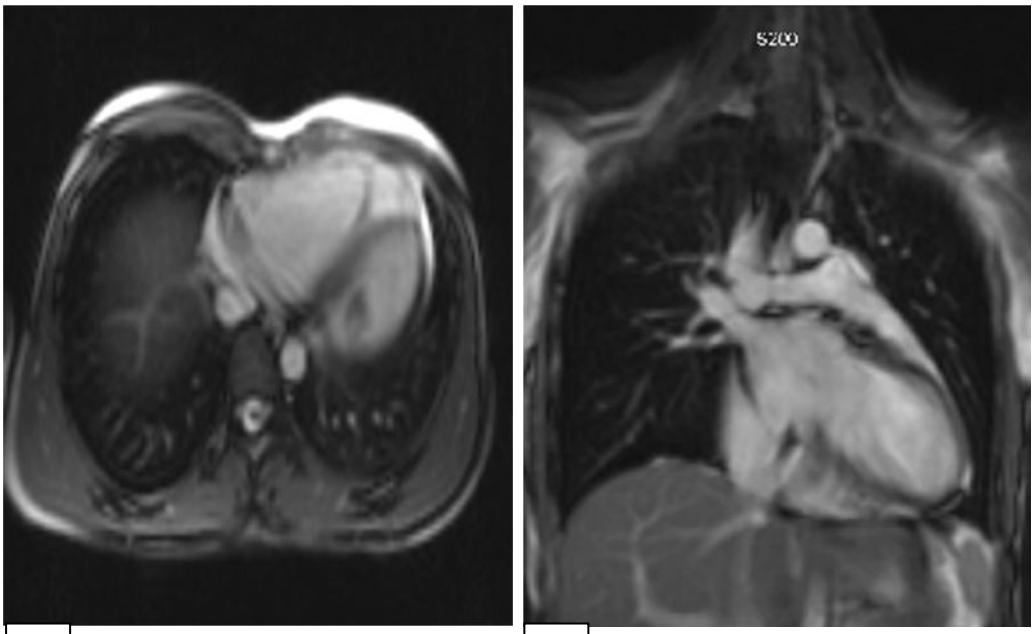
b) MRI coils

Coil 16 channel Body Array Anterior coil was used

c) MRI protocol – Sequences and Technique

The cardiac MRI for viability protocols included the following sequences –

A. 2D TRUFISP axial (transverse) section of the chest (FOV - 400 mm; slice thickness - 6 mm with slice gap of 1.2 mm; Repetition time TR – 161.83 ms; Echo time TE – 1.17 ms; matrix size 136x256; Acquisition window 850; Trigger pulse 1; Trigger delay 688; One acquisition scan time approximately 50 seconds in an ideal situation; scan time depends partly on patients ability to cooperate in the given situation; concatenations vary depending on patient's condition).



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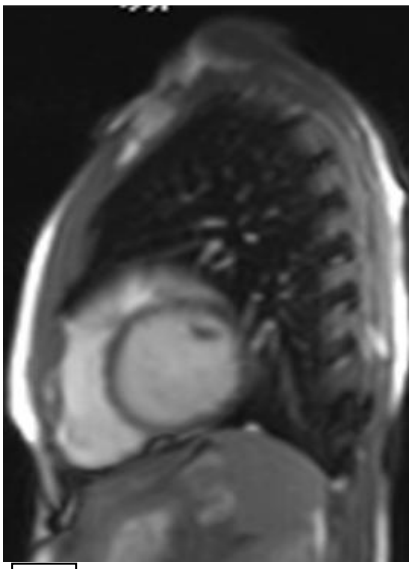
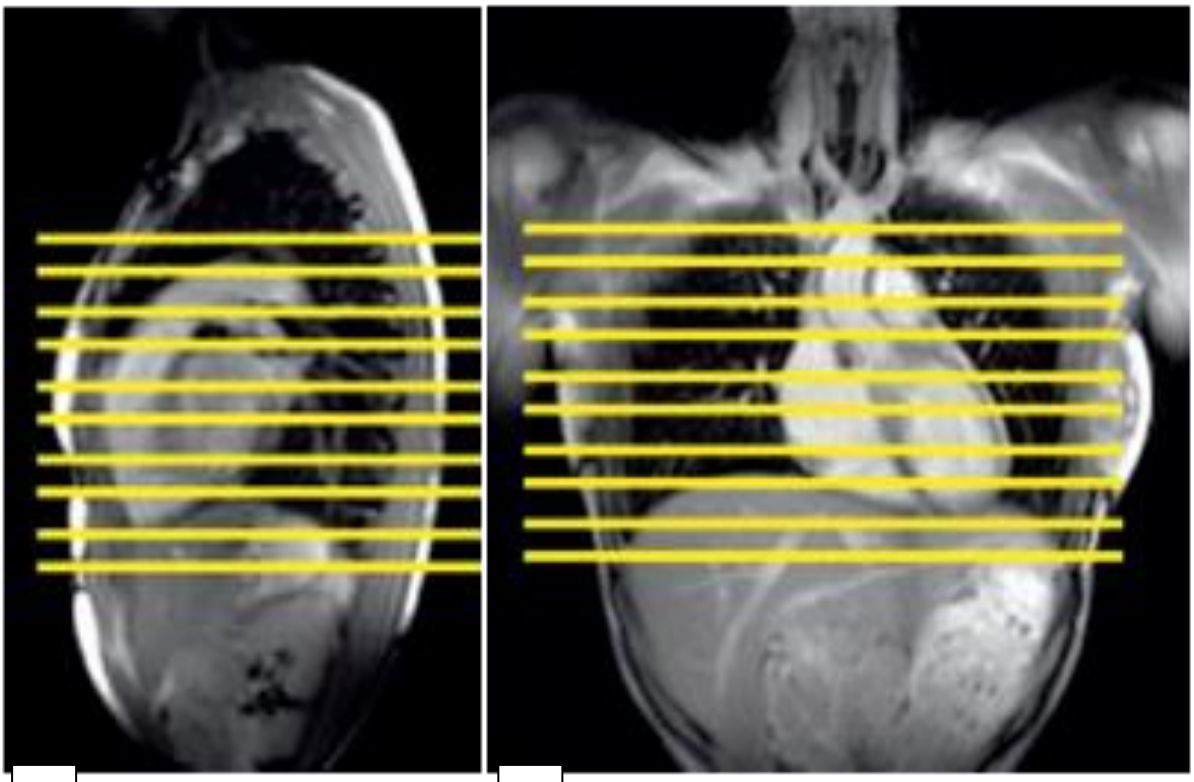


c

Fig 10 a, b, c TRUFISP scout images for planning localizers in axial, coronal and sagittal sections

On this TRUFISP axial image, the localizer for two chamber is planned. On the two chamber image thus acquired, the four chamber localizer is planned. On the four chamber image thus acquired, the short axis localizer is planned. The true four chamber cine is now planned on the short axis image acquired.



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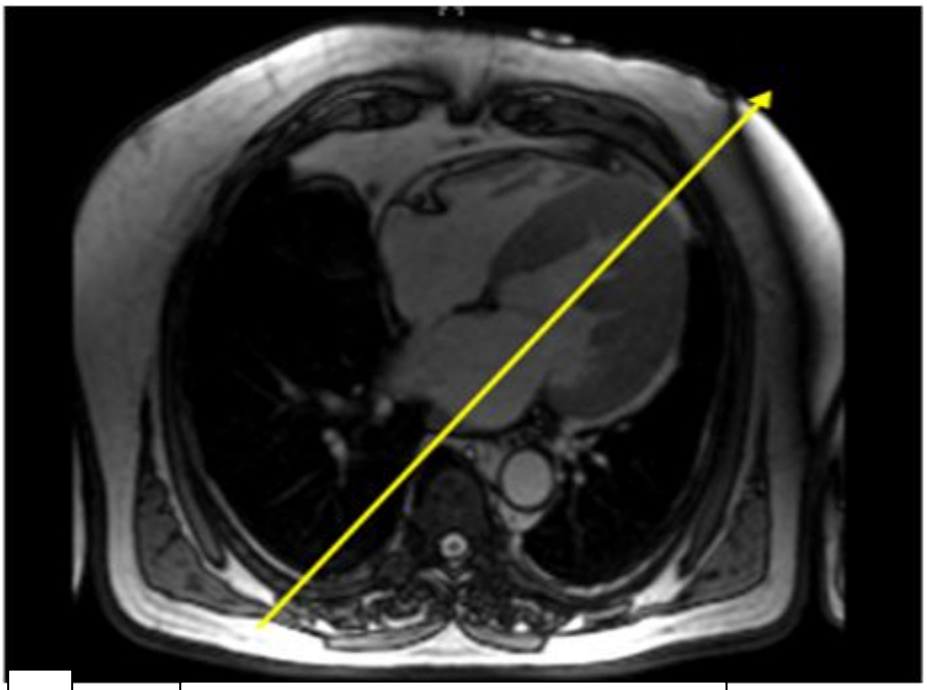
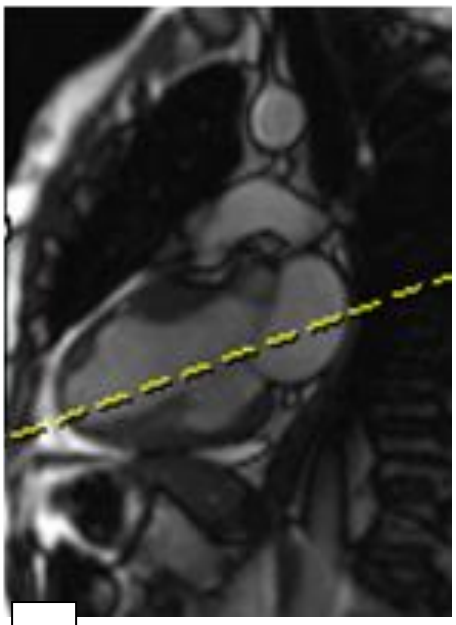
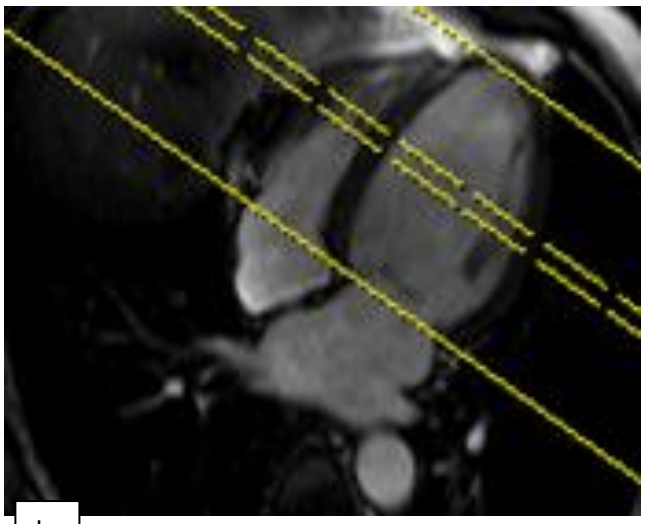


c

Fig 11 a,b, c Planning for localizers



a



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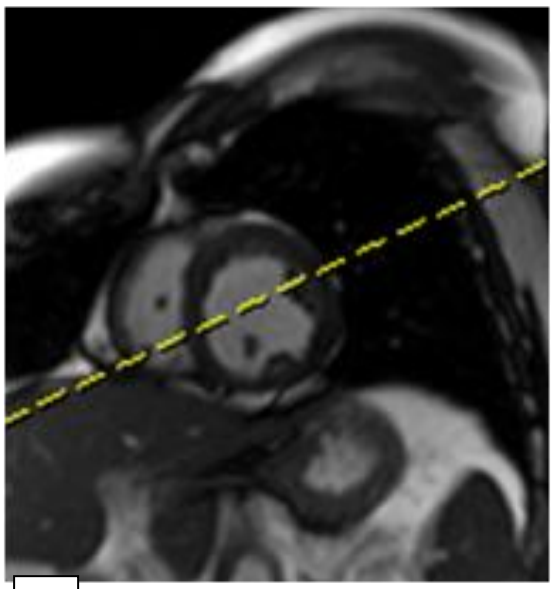

c

Fig 12 a,b,c Planning on localizers

B. TRUEFISP four chamber cine (FOV – 350; slice thickness – 6 mm; slice gap 1.2 mm; Repetition time TR – 39.15; Echo time TE - 1.12; matrix size – 156x192; Flip angle – 66; Dynamic measurement – 1; physio – cine – on; No fat suppression; Calculated phases – 25; One acquisition scan time – 15 seconds.

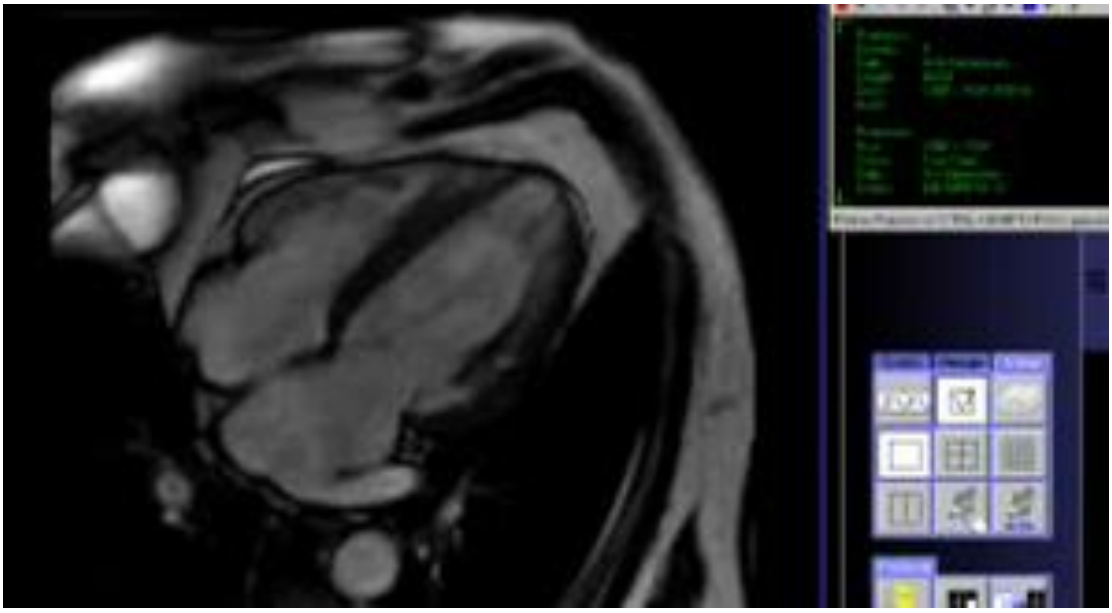

Fig 13 True four chamber

On the true four chamber cine image, the true two chamber image of the left and right heart are acquired.

C. TRUEFISP two chamber cine of the left heart (FOV – 350; slice thickness – 6 mm; slice gap 1.2 mm; Repetition time TR – 39.15; Echo time TE - 1.12; matrix size – 156x192; Flip angle – 66; Dynamic measurement – 1; physio – cine – on; No fat suppression; Calculated phases – 25; one acquisition scan time – 15 seconds).

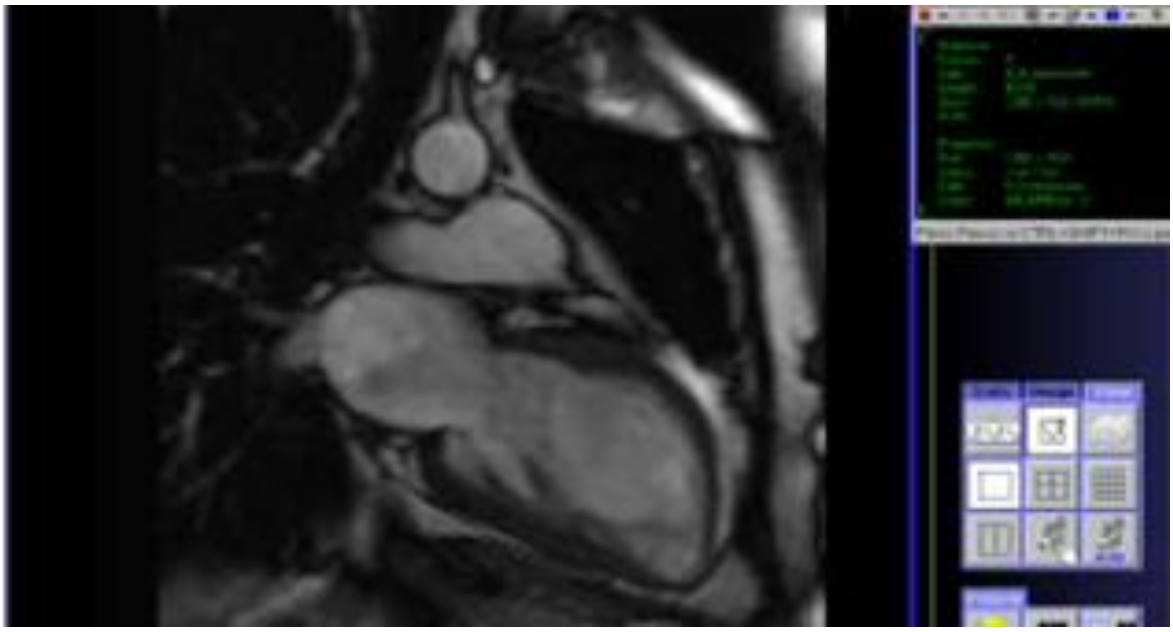

Fig 14 True two chamber

D. TRUEFISP two chamber cine of the right heart (FOV – 350; slice thickness – 6 mm; slice gap 1.2 mm; Repetition time TR – 39.15; Echo time TE - 1.12; matrix size – 156x192; Flip angle – 66; Dynamic measurement – 1; physio – cine – on; No fat suppression; Calculated phases – 25; one acquisition scan time – 15 seconds).

E. TRUFISP Short axis cine: The short axis cine is planned on the true four chamber cine image. It is acquired from base to apex. (FOV 350; Slice thickness – 6 mm; slice gap 1.2 mm; no of slices – 12; Repetition time TR – 57.88; Echo time TE – 1.12; Flip angle – 80; physio – cine – on; calculated phases 25; concatinations -12; segments – 22; one acquisition scan time – 1.36 seconds).

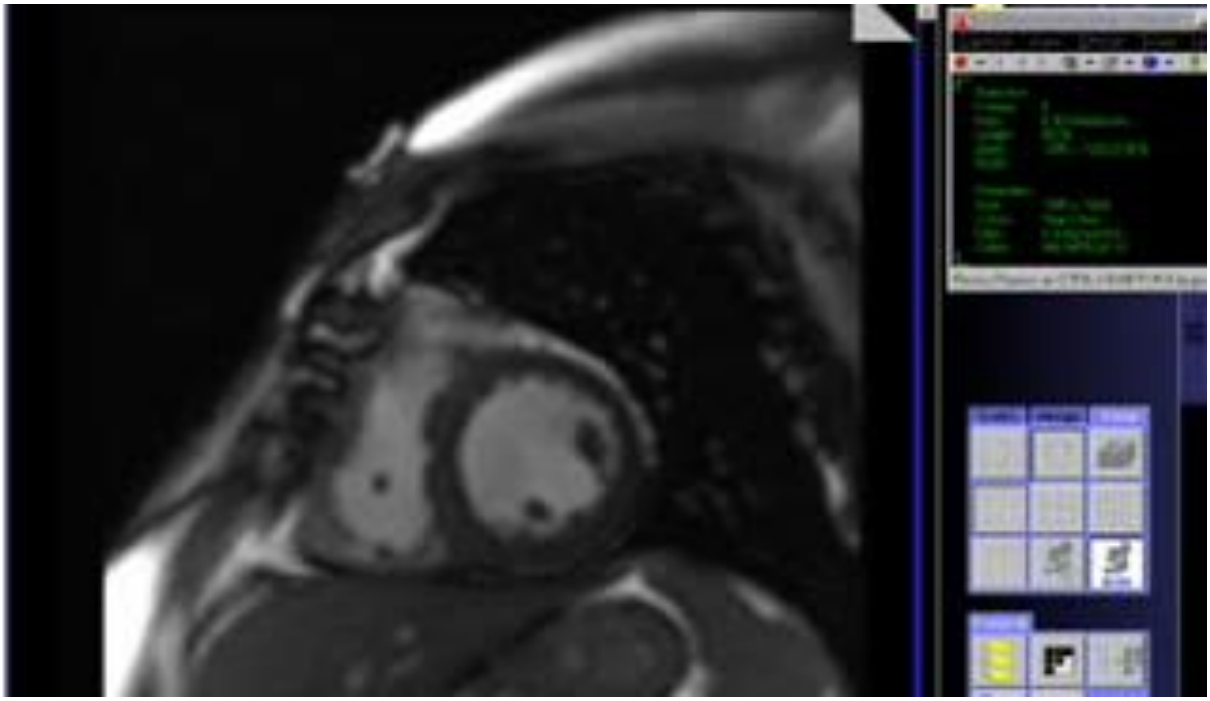

Fig 15 True short axis

Left Ventricle - Absolute				
Cardiac Function			Normal Range (F) (MRI)	Units
Ejection Fraction	EF	65.5	56.00 ... 78.00	%
End Diastolic Volume	EDV	53.1	52.00 ... 141.00	ml
End Systolic Volume	ESV	18.3	13.00 ... 51.00	ml
Stroke Volume	SV	34.8	33.00 ... 97.00	ml
Cardiac Output	CO	2.50	2.65 ... 5.98	l/min

Fig 16 Left ventricular function generated by Syngo workstation

15 ml of gadolinium DTPA is injected over approximately 1 minute. Perfusion scans are performed in the short axis view.

F. PERFUSION in short axis images. (FOV – 360; slice thickness – 8; slice gap – 10 mm; no of slices – 4 mm; cine – off; Repetition time TR – 173.78; Echo time TE – 1.14; Time of inversion TI – 100; one acquisition scan time 1:58).

G. TI scout (FOV – 350; Repetition time TR – 23.49; Time of echo TE – 1.12; Magnitude preparation on TI scout; Flip angle - 30)

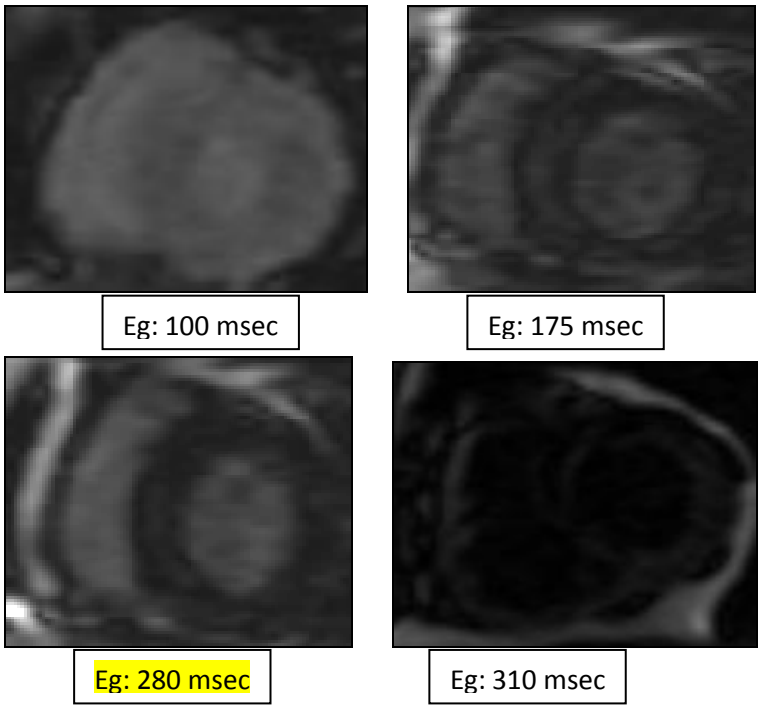

Fig 17 - Time of inversion scout: is required to choose the time of inversion at which the myocardium and blood contrast is optimal before the viability scan in PSIR sequence. The TI can range from 175 to 300

H. Viability scanning Delayed imaging done with viability scanning at 10 minutes post contrast injection. (FOV – 350; slice thickness – 6 mm; slice gap 1.2 mm; Repetition time TR – 39.15; Echo time TE - 1.12; matrix size – 156x192; Flip angle – 66; Dynamic measurement – 1; physio – cine – on; No fat suppression; Calculated phases – 25; one acquisition scan time – 15 seconds)

INTERPRETATION OF ECG, ECHO AND CORONARY ANGIOGRAPHY

The electrocardiography, echocardiography and coronary angiography of a recruited patient was interpreted and reported by the co investigator of the same unit under which the patient was being treated, in a format designed for the study. The archived ECHO images on the ultrasound machine in the cardiology department was reviewed and reported in the provided format. The coronary angiography was also reviewed by the same co-investigator .

INTERPRETATION OF CARDIAC MRI:

17 cardiac segment model as described by the American Heart Association was used in the description of the extent of both the hyperenhancement and wall motion abnormality or any other lesion that may be identified

An area of ischemic myocardium showed up as a regional wall motion abnormality in the cine MR images. The infarcted tissues showed up as segments hyperenhancement of variable transmurality on the post gadolinium-DTPA delayed scans.

Left ventricular function: The LV ejection fraction was calculated by the Siemens 1.5 T MRI machine specific software which is a dedicated software provided by the same company. The software used is Argus software (Siemens, Erlangen Germany AG) .This software is already validated, being used widely internationally and is approved by the SCMR (Society for Cardiac Magnetic Resonance Imaging)

The software allows us to outline the endocardium in multiple planes of short axis view of both the ventricles at end diasotole and end systole. The machine then generates the ejection fraction, stroke volume and cardiac output from this.

Wall motion abnormality was scored both qualitatively and quantitatively:

Qualitatively by visual eyeballing

0 – normal

1 - mild or moderate hypokinesia

2 - severe hypokinesia

3 - akinesia, and

4 – dyskinesia

Quantitative wall motion abnormality will be scored by the formula: (end-systolic wall thickness) - (end-diastolic wall thickness) * 100 / end-diastolic wall thickness

Contractility based on % wall thickness:

>30% wall thickness indicates normal

20-29% wall thickness indicates mild hypokinesia

10-19 % wall thickness indicates moderate hypokinesia

<10 % wall thickness indicates severe hypokinesia

Enhancement pattern was be scored as :

0 – 0% Hyperenhancement in a RWMA

1 - Hyperenhancement of 1 to 25% thickness of the wall in a myocardium with RWMA

2 - Hyperenhancement of 26 to 50% thickness of the wall in a myocardium with RWMA

3 - Hyperenhancement of 51 to 75% thickness of the wall in a myocardium with RWMA

4 - Hyperenhancement of 76 to 100% thickness of the wall in a myocardium with RWMA

The following conclusions were made for the extent of mural delayed hyperenhancement of the myocardium

In segments showing 1-25% of extension of delayed hyperenhancement (representing infarct), 65% show improved contractility after revascularization.

In segments showing 26-50% of extension of delayed hyperenhancement (representing infarct), 43% show improved contractility after revascularization.

In segments showing 51-75% of extension of delayed hyperenhancement (representing infarct), 10% show improved contractility after revascularization.

In segments showing 76-100% of extension of delayed hyperenhancement (representing infarct), 0% show improved contractility after revascularization.

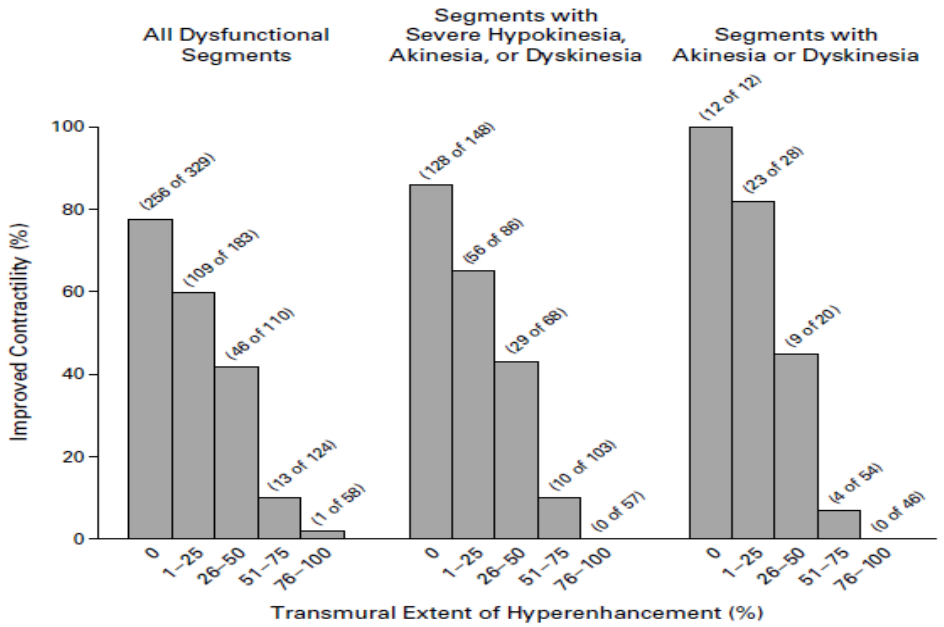

Figure 4. Relation between the Transmural Extent of Hyperenhancement before Revascularization and the Likelihood of Increased Contractility after Revascularization.

Data are shown for all 804 dysfunctional segments and separately for the 462 segments with at least severe hypokinesia and the 160 segments with akinesia or dyskinesia before revascularization. For all three analyses, there was an inverse relation between the transmural extent of hyperenhancement and the likelihood of improvement in contractility.

Fig 18 - Courtesy Kim et al(20)

INSTITUTIONAL REVIEW BOARD APPROVAL AND FUNDING :

Institutional review board (IRB) approval was obtained prior to the commencement of the study (IRB minutes number 8231 dated 19.03.2013).

STATISTICAL ANALYSIS:

Statistical analyses were performed using SPSS software version 18.

1) Analysis of the presence of infarct identified on ECG and MRI

2) Analysis of the regional wall motion abnormality scored quantitatively and objectively in MRI cine images.

3) Analysis of the regional wall motion abnormality assessed objectively in echocardiography and in MRI cine images.

4) Analysis of the regional wall motion scored quantitatively in MRI cine images and in echocardiography.

5) Analysis of wall motion abnormality as seen in MRI cine images and the degree of transmural enhancement in post contrast studies.

6) Analysis of wall motion abnormality as seen in echocardiography and the degree of transmural enhancement in post contrast studies.

RESULTS

A total number of 340 segments were studied. Of this total 155 segments were diseased. The total number of patients who participated in the study was 20.

DEMOGRAPHY

A. Age:

Of the total number of patients recruited, the mean age was around 56 years. Average age for males was 57.2 years and the average age for females was 52.6 years. 50% of the study population were in the age group of 50 to 60 years, 10% each were in the age group of 30 to 50 and 70 to 80 years, 20% were in the age group of 60 to 70 years.

{Fig 19.1, table 1}

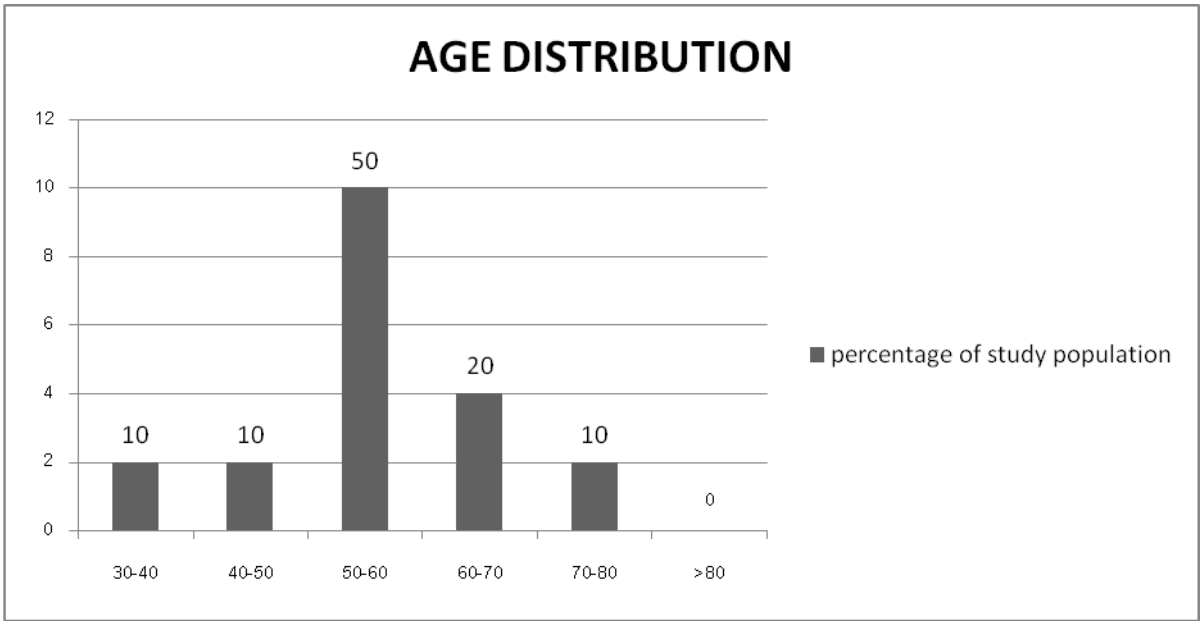

Fig. 19.1 – Distribution of age in study population

Table 1- Age distribution in population

Age groups (Years)	**Numbers**	**Percentages**
30 – 40	2	10%
40 – 50	2	10%
50 – 60	10	50%
60 – 70	4	20%
70 – 80	2	10%
>80	0	0%

B. Gender:

85% (17) of the population were male. Only 15% (3) of the study population were female {Fig19.2}

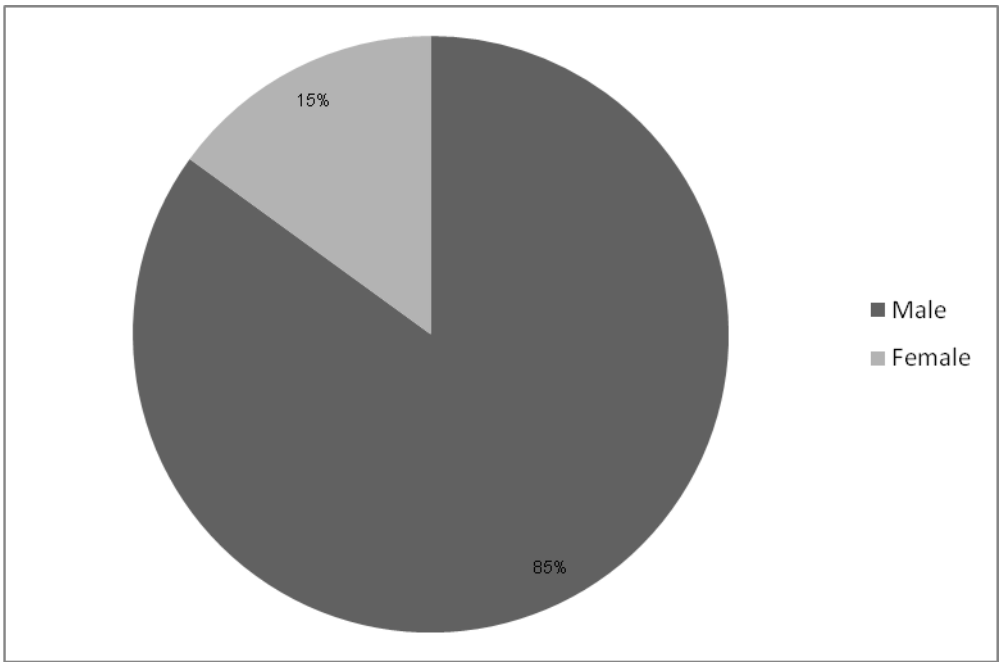

Fig 19.2 Gender wise distribution of the study population

C. Geographic distribution of study population:

35% (7) of the patients participating in the study were from West Bengal, 25% (5) were from Bihar and another 25% (5) from Tamil Nadu, 10% (2) were from Bangladesh and 5% (1) were from Kerela. {Fig 19.3, table 2}

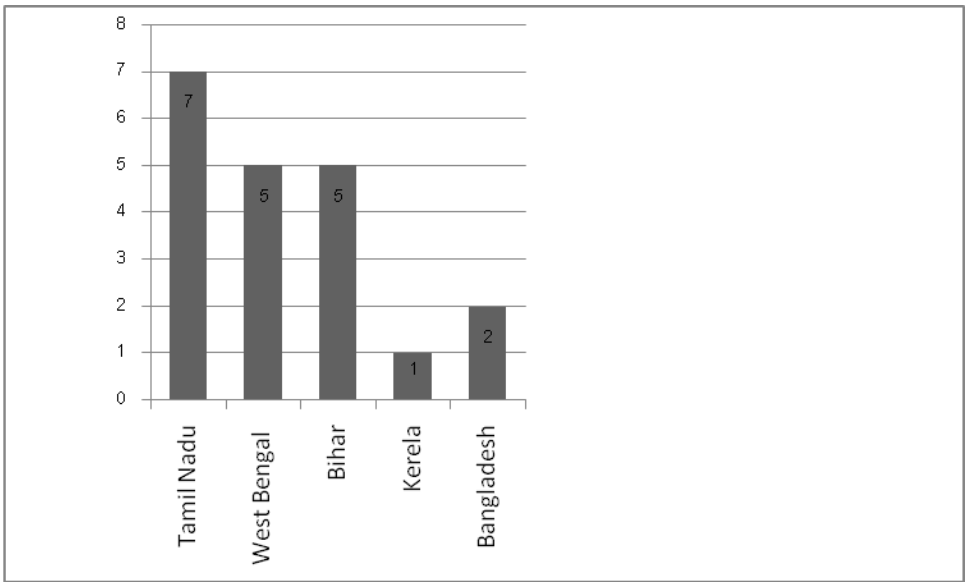

Fig. 19.3 Geographic distribution of the study population

Table 2- Geographic (state wise) distribution of the study population

Region	Number	Percentage
Bangladesh	2	10
Bihar	5	25
Kerela	1	5
Tamil Nadu	7	35
West Bengal	5	25

CLINICAL FEATURES:

A. Diabetes mellitus: 55% of the study population had diabetes mellitus and 45% were non diabetic. {Table 3}

Table 3- Prevelence of diabetes among the study population as a risk factor to coronary artery disease.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Notdiabetic	9	45.0	45.0	45.0
	Diabetic	11	55.0	55.0	100.0
	Total	20	100.0	100.0	

B. Hypertension: Exactly 50% of the study population were hypertensive. {Table 4}

Table 4- Prevelence of hypertension among the study population as a risk factor to coronary artery disease.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Nothypertensive	10	50.0	50.0	50.0
	Hypertensive	10	50.0	50.0	100.0
	Total	20	100.0	100.0	

C. Dyslipidemia: 25% of our study population had dyslipidemia. 75% of the study population did not have dyslipidemia. {Table 5}

Table 5- Prevelence of dyslipidemia among the study population as a risk factor to coronary artery disease.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	nodyslipidemia	15	75.0	75.0	75.0
	dyslipidemia	5	25.0	25.0	100.0
	Total	20	100.0	100.0	

D. Obesity: 40% of the study population were obese, 5% had normal BMI appropriate for height. There was no information regarding 50% of the study population. {Table 6}

Table 6- Prevalence of obesity among the study population as a risk factor to coronary artery disease.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	notobese	1	5.0	5.3	47.4
	Obese	8	40.0	42.1	42.1
	noinfo	10	50.0	52.6	100.0
	Total	19	95.0	100.0	
Missing	System	1	5.0		
Total		20	100.0		

E. Smoking: 35% of our study population gave history of smoking, 35% were non smokers, there was no information available for the rest 30% of the study population. {Table 7}

Table 7- Prevalence of smoking among the study population as a risk factor to coronary artery disease.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	nonsmoker	7	35.0	35.0	35.0
	Smoker	7	35.0	35.0	70.0
	Noinfo	6	30.0	30.0	100.0
	Total	20	100.0	100.0	

F. Lifestyle: 15 % of our study population had sedentary lifestyle, 35% had non sedentary lifestyle and there was no information available for the rest of the 50% of the study population. {Table 8}

Table 8- Lifestyle among the study population as a risk factor to coronary artery disease.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	nonsedentary	7	35.0	35.0	35.0
	sedentarylifestyle	3	15.0	15.0	50.0
	Noinfo	10	50.0	50.0	100.0
	Total	20	100.0	100.0	

G. Level of stress: 35% of our study population gave history of stressful jobs / work, 20%gave history of non stressful jobs / work. No information was available for the rest of the 45% of the population. {Table 9}

Table 9- Presence of stressful environment among the study population as a risk factor to coronary artery disease.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Nostress	4	20.0	20.0	55.0
	Stress	7	35.0	35.0	35.0
	Noinfo	9	45.0	45.0	100.0
	Total	20	100.0	100.0	

H. History of angina: 65% of our study population gave history of prior angina attacks. 30% did not have prior angina. No information available in 5% of the study population. {Table 10}

Table 10- History of angina among the study population.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Nohistoryofangina	6	30.0	30.0	30.0
	Historyofangina	13	65.0	65.0	95.0
	Noinfo	1	5.0	5.0	100.0
	Total	20	100.0	100.0	

FINDINGS ON ECG, ECHOCARDIOGRAPHY AND CORONARY ANGIOGRAPHY

A. Infarct as identified by ECG : Out of the 20 ECG studied, 19 (95%) showed evidence of prior ischemic insult. Only 1 (5%) ECG showed non specific findings. {Fig 20.1}

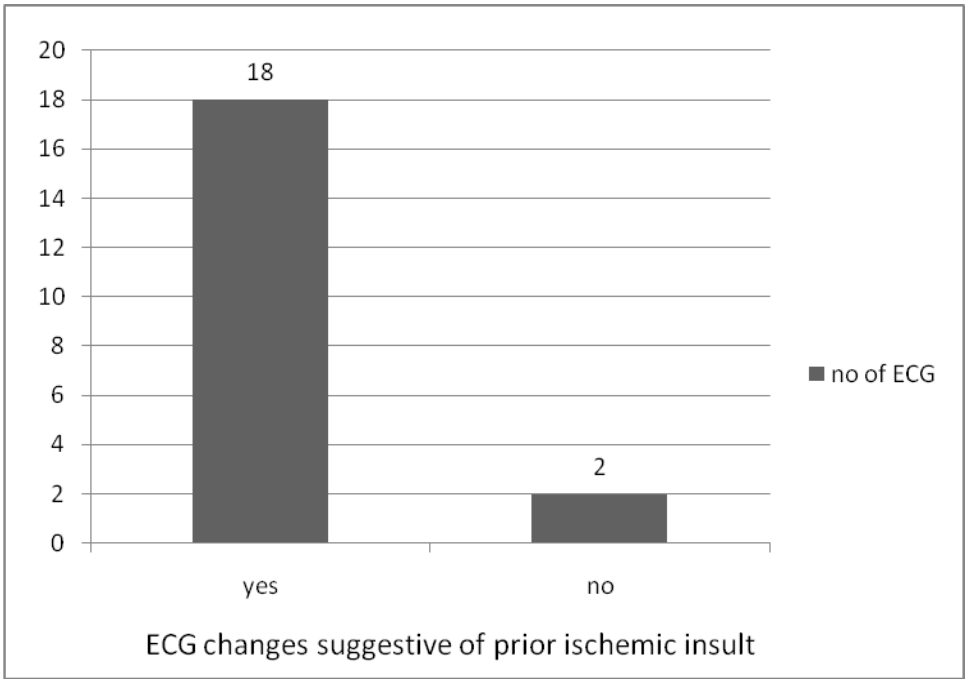

Fig 20.1 Presence of evidence of prior ischemic insult in the ECG of the given patients in the study population

B. Wall motion abnormality as identified by echocardiography:

Out of the 340 segments that were studied by visual assessment on echocardiography, 177 segments were found to have wall motion abnormality. Out of the 177 segments, 158 segments (89.2%) were found to be mild to moderate hypokinetic, 11 segments (6.2%) were found to be severely hypokinetic and 8 segments (4.5%) were found be to be akinetic / dyskinetic. {Fig 20.2}

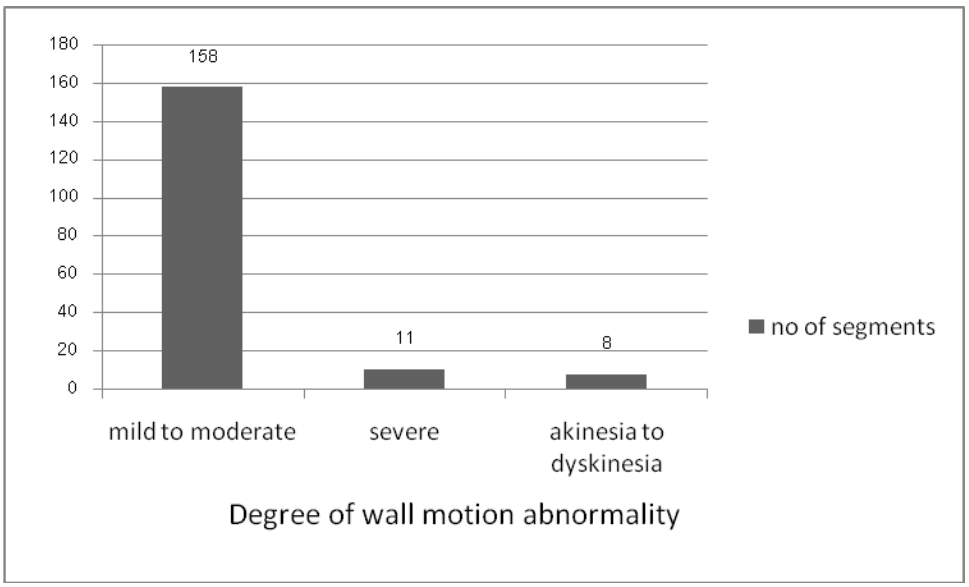

Fig 20.2 Degree of wall motion abnormality in diseased segments of the myocardium as assessed visually ("eye balling") on Echocardiography.

C. Left ventricular ejection function on ECHO:

In our study population, 40% (8) had ejection fraction between 30-40% and another 40% (8) had ejection fraction between 40 and 50%. 2 patients (10%) had ejection fraction between 20-30 and another 2 patients (10%) had ejection fraction of 50-60%. {Fig 20.3}

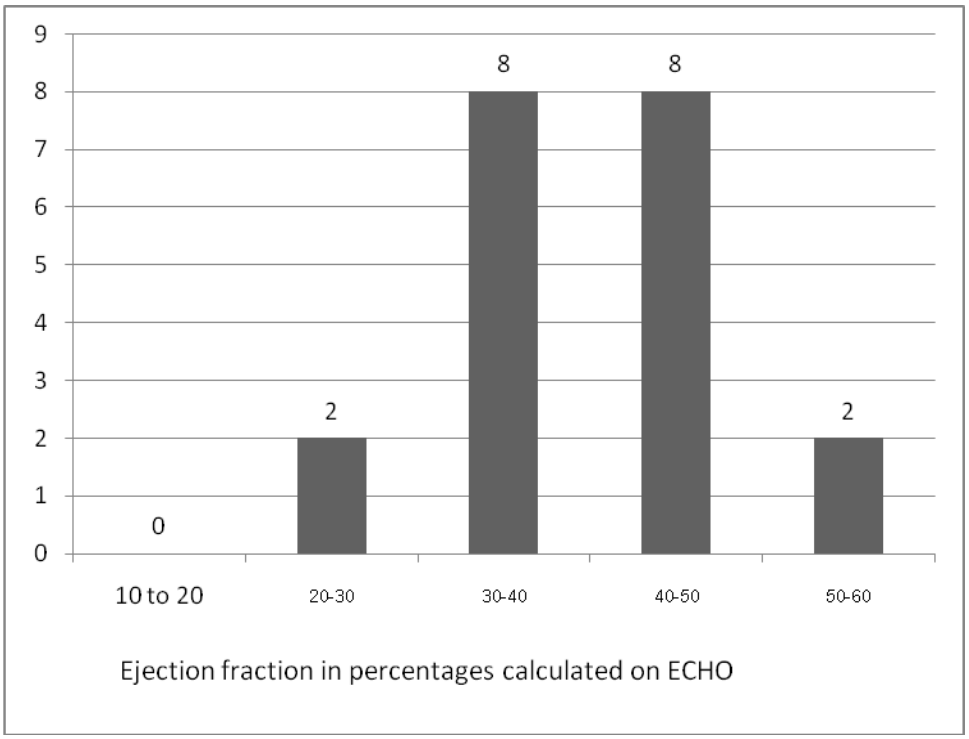

Fig 20.3 Ejection fractions calculated on echocardiography

D. Diseased coronary vessels as assessed by coronary angiography:

Out of the total number of 60 vessels studied, 38 vessels were diseased.

Out of the 38 vessels diseased, 14 vessels (36.8%) were LAD artery, 12 vessels each (31.5% each) were the LCx artery and the RCA.

Of the total of 14 LAD arteries showing disease, 13 vessels (92.8%) had significant occlusion and 1 (7.1%) vessel had intermediate occlusion. Of the total of 12 LCx arteries showing

disease, 8 vessels (66.6%) had significant occlusion and 4 vessels (33.3%) had intermediate occlusion. And of the total of 12 RCA showing disease, 10 vessels (83.3%) had significant occlusion, 1 (8.3%) vessel had intermediate occlusion and 1 (8.3%) other vessel had mild disease. {Fig 20.4}

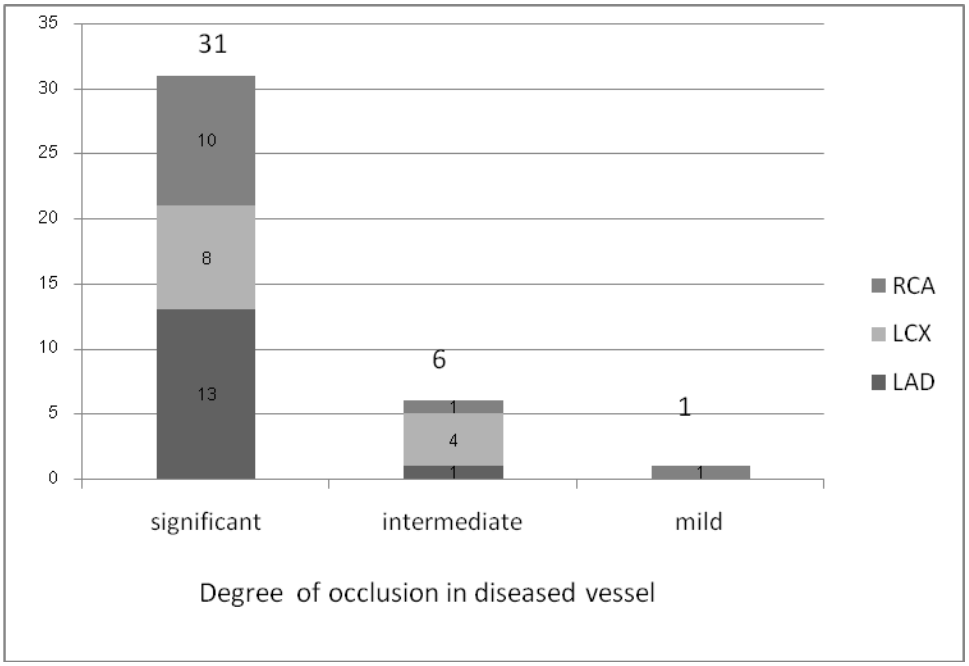

Fig 20.4 Degree of occlusion of the diseased vessels identified on coronary angiography. <50% occlusion is classified as mild; 50-70% occlusion as intermediate; >70% occlusion as significant occlusion.

FINDINGS ON CARDIAC M RI:

A. Degree of wall motion abnormality (Method 1, as assessed visually) of diseased segments:

Out of 340 segments studied by visual assessment of myocardial wall motion contractility, 177 segments were found to have wall motion abnormality. Out of the 177 segments, 78 segments (40%) were found to be akinetic / dyskinetic. 59 segments (33.3%) were found to be severely hypokinetic and 40 segments (22.6%) were found to be mild to moderately hypokinetic. {Fig 20.5}

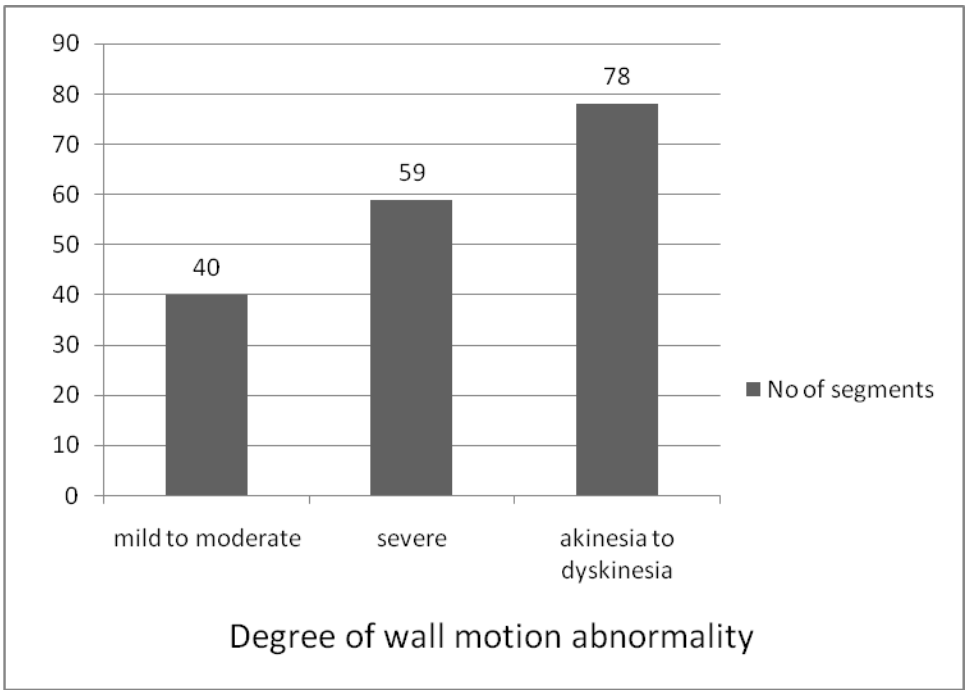

Fig 20.5 Degree of wall motion abnormality in diseased segments of the myocardium as assessed visually ("eye balling") on cine MRI.

B. Degree of wall motion abnormality (Method 2, as assessed by wall thickness measurement at end systole and diastole) of diseased segments:

Out of 340 segments studied for wall motion abnormality by measurement of wall thickness at systole and diastole, 158 segments were found to have wall motion abnormality. Out of the 158 segments, 73 segments (46.2%%) were found to be akinetic / dyskinetic. 60 segments (37.9%) were found to be severely hypokinetic and 25 segments (15.8%) were found to be mild to moderately hypokinetic. {Fig 20.6}

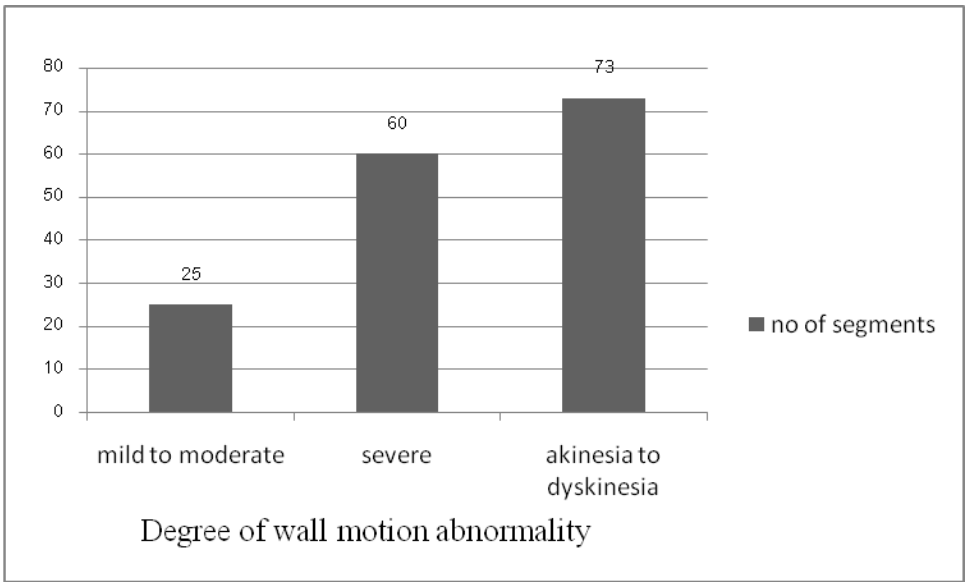

Fig 20.6 Degree of wall motion abnormality in diseased segments of the myocardium as assessed by measurement of wall thickness at end diastole and end systole on cine MRI.

C. Left ventricular function measured by cine SA MRI images:

40% of the study population showed ejection fraction between 20 to 30% when calculated by MRI short axis cine images. 25% (5) showed ejection fraction between 40 to 50%, 20% (4) showed ejection fraction between 10-20%, 10% (2) showed ejection fraction between 30-40% and 5% (1) showed ejection fraction between 50-60%. {Fig 20.7}

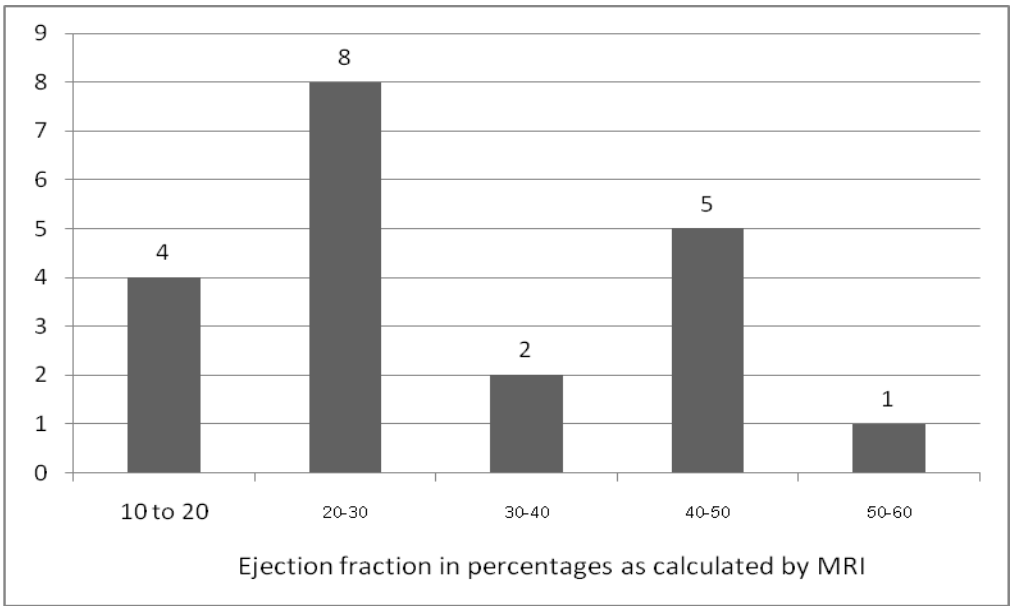

Fig 20.7 Left ventricular ejection fraction as measured by cardiac MRI

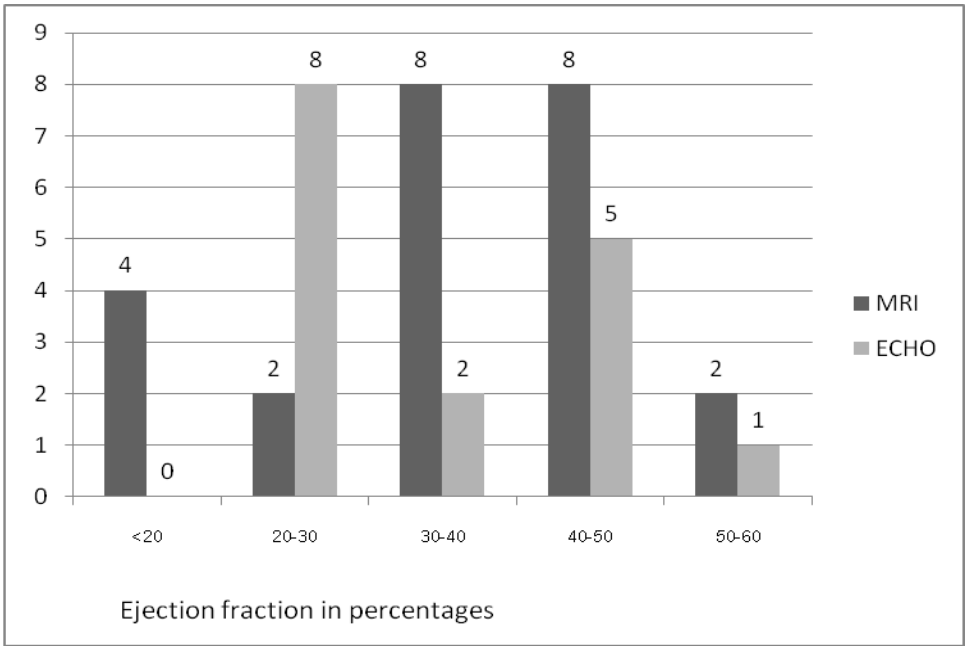

Fig 20.8 Left ejection fraction as measured by echocardiography and MRI

D. Degree of delayed enhancement of diseased segments:

Out of 340 segments studied, 155 segments were found to be infarcted by delayed post contrast scan in MRI in the form of the myocardium showing delayed enhancement. Of the 155 segments 120 segments (77.4%) showed 75 - 100% (transmural) of myocardial enhancement on delayed enhancement scan. 24 segments (15.4%) showed 26 – 50% of myocardial enhancement, 6 segments (3.8%) showed 5- - 75% of myocardial enhancement and 5 segments (3.2%) showed 1 – 25% of myocardial enhancement. {Fig 20.9}

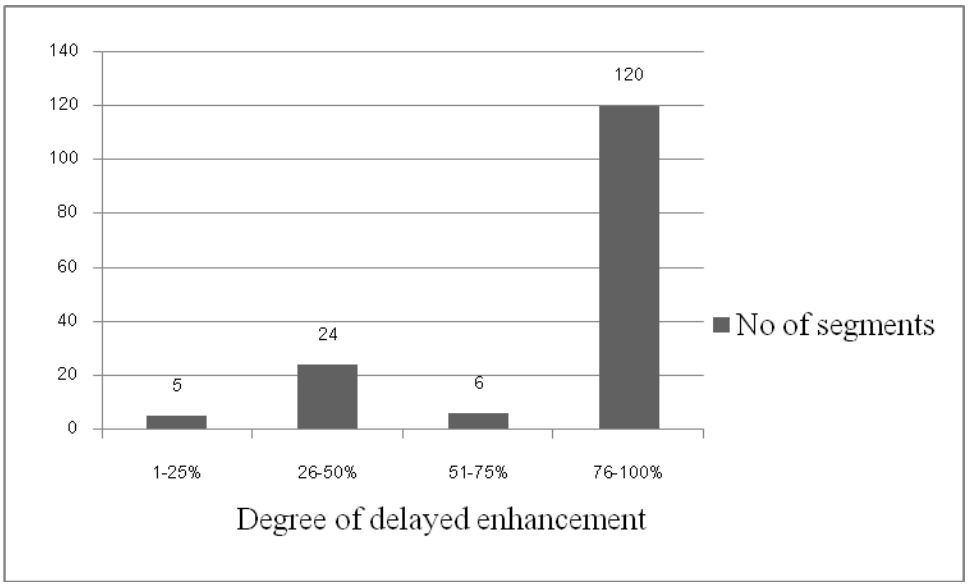

Fig 20.9 Degree of enhancement or the transmurality of delayed enhancement in diseased segments of the myocardium on delayed PSIR sequence of cardiac MRI.

STATISTICAL ANALYSIS:

A. Intra class correlation between left ventricular ejection fraction measured by ECHO and that measured by Short axis view of the left ventricle on MRI. {Table 11.1}

Table 11.1- Table showing Intra class correlation of the performance of ECHO and MRI in estimation of the left ventricular ejection fraction.

	Intraclass Correlation[a]	95% Confidence Interval		F Test with True Value 0			
		Lower Bound	Upper Bound	Value	df1	df2	Sig
Single Measures	.555[b]	.161	.797	3.495	19	19	.005
Average Measures	.714[c]	.277	.887	3.495	19	19	.005

N=20; r 0.555 and p<=0.005

B. Pearson's Correlation between left ventricular ejection fraction measured by ECHO and that measured by Short axis view of the left ventricle on MRI. {Table 11.2}

Table 11.2- Table showing Pearson's correlation of the performance of ECHO and MRI in estimation of the left ventricular ejection fraction.

		ECHO EF for LV function on ECHO	MRI EF for LV function on MRI
ECHO EF for LV function on ECHO	Pearson Correlation	1	.602**
	Sig. (2-tailed)		.005
	N	20	20
MRI EF for LV function on MRI	Pearson Correlation	.602**	1
	Sig. (2-tailed)	.005	
	N	20	20

n- 20; r- 0.602; p<=0.005

C. Scatter plot showing correlation between the left ventricular ejection fraction measured by ECHO and MRI. {Fig21.1}

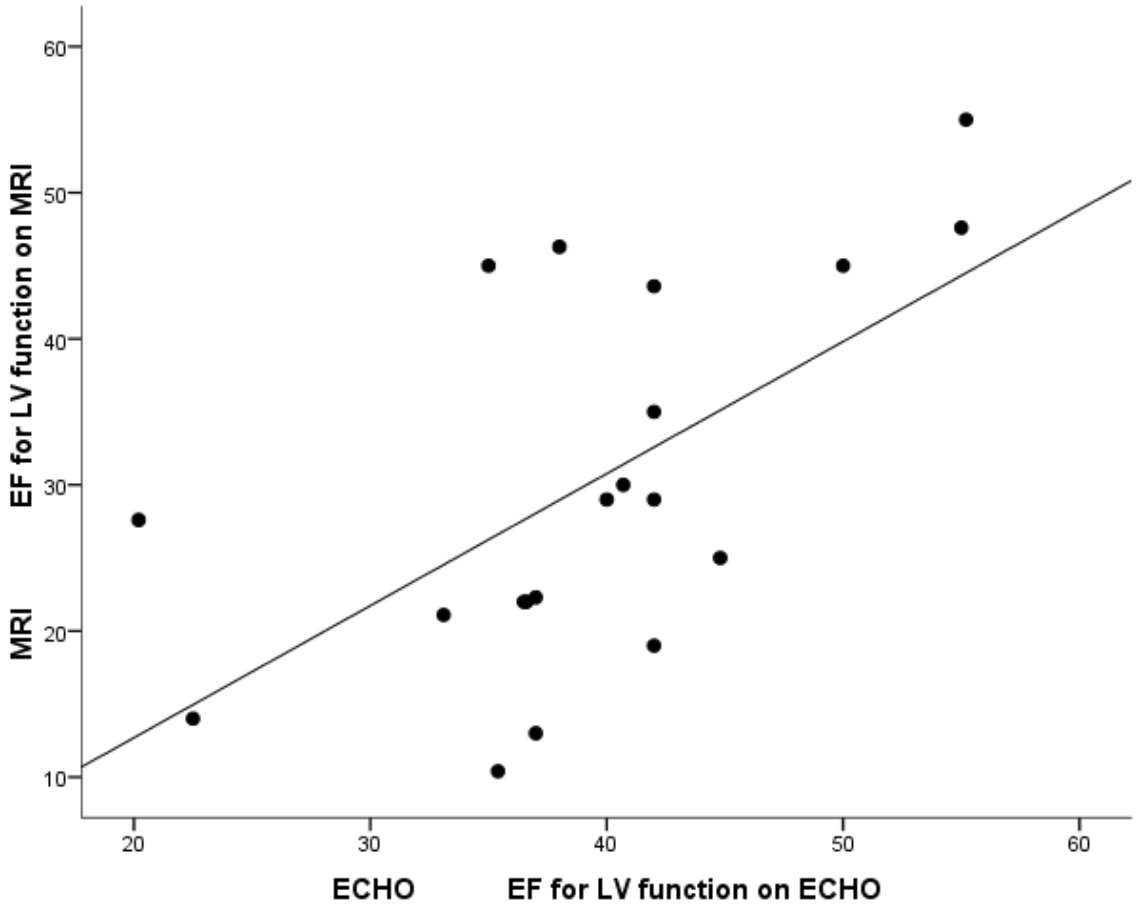

Fig 21.1 Correlation between ejection fraction measured by ECHO and MRI. N-20; r- .602 ; p<=0.005

D. BLAND ALTMAN PLOT {Fig 21.2}

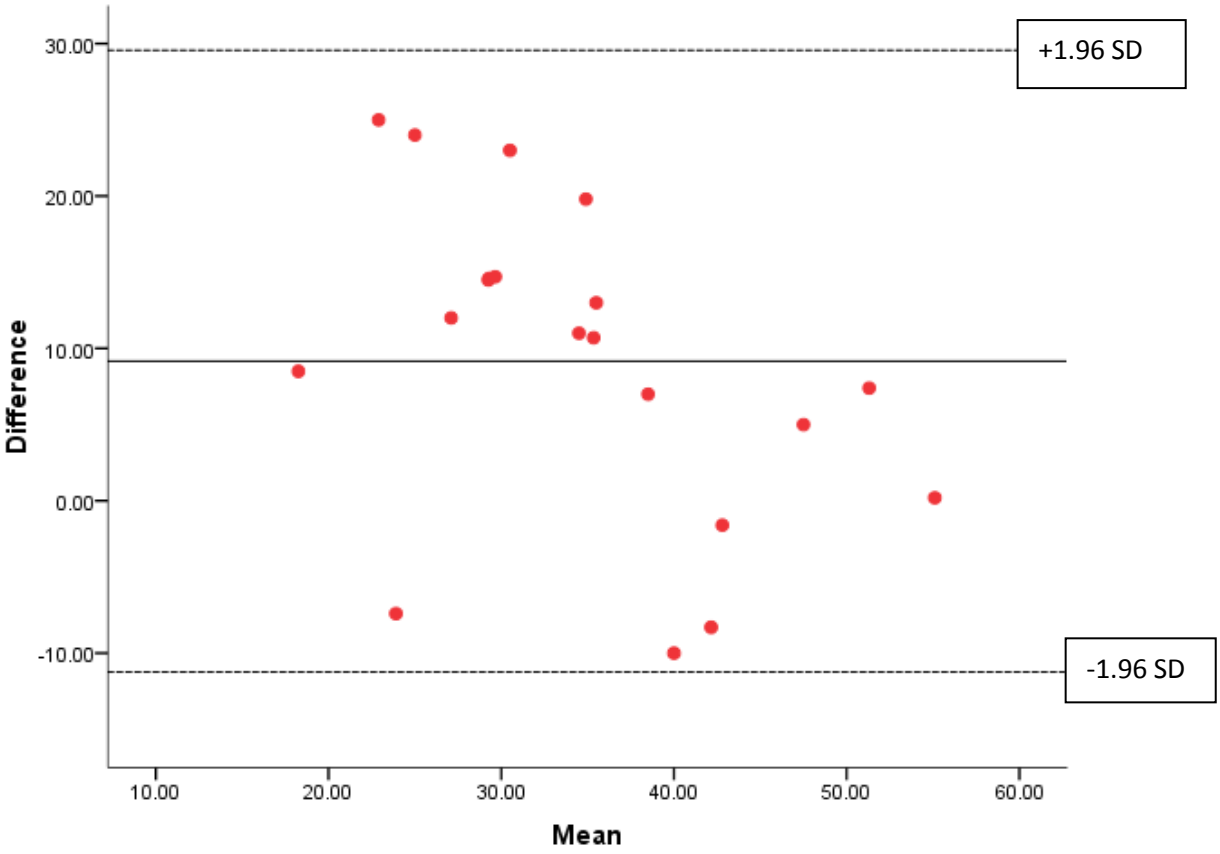

Fig 21.2 – Differences between ejection fraction of the left ventrical when measured by ECHO and MRI based on the Bland- Altman plot. The central continuous horizontal line is the mean difference whereas the area between the two interrupted lines represents the area of 95% agreement limit. Below is histogram showing normal distribution curve of the differences. {21.3}

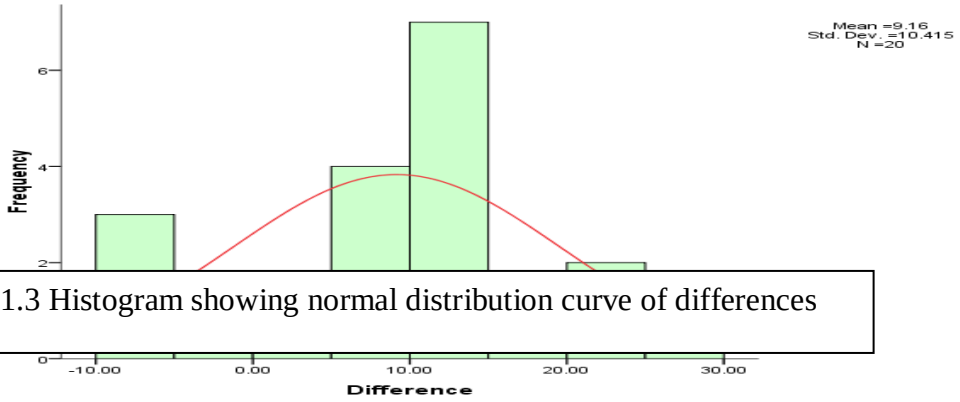

Fig 21.3 Histogram showing normal distribution curve of differences

E. Wall motion assessed in cardiac MRI cine images by two methods: wall thickness measurement method and visually by eye balling {Fig 21.4}

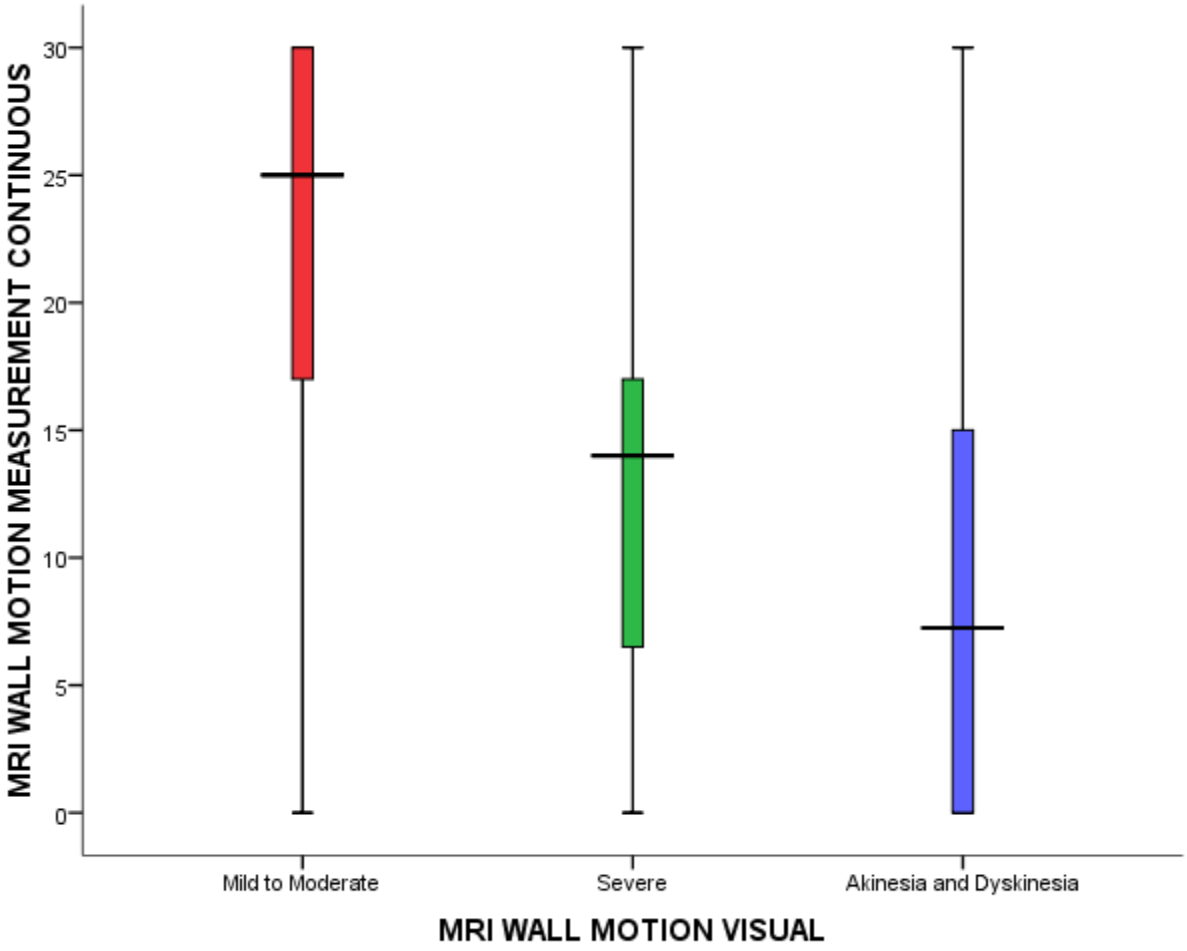

Fig 21.4 Box and whisker plot showing the wall motion abnormality assessed by wall thickness measurement and visually by eyeballing on cine MRI images

F. Agreement between coronary vessel being diseased in coronary angiogram and infarct in corresponding segment in MRI {Table 11.3}

Table 11.3 Kappa agreement test for coronary disease in angiogram and infarct in corresponding segment in MRI

		Value	Asymp Std Error	Approx T	Approx Sig
LAD	Measure of Agreement Kappa No of valid cases	.294 20	.222	1.353	.176
LCX	Measure of Agreement Kappa No of valid cases	.375 20	.211	1.677	.094
RCA	Measure of Agreement Kappa No of valid cases	-.023 20	.221	-102	.919

G. Association between percentage of occlusion of coronary artery and corresponding segmental transmural enhancement {Table 11.4}

Table 11.4 Chi square test between % of occlusion and extent of transmural delayed enhancement

		Value	Asymp Sig
LAD	Measure of Agreement Kappa	9.451	.024
	No of valid cases	20	
LCX	Measure of Agreement Kappa	3.450	.751
	No of valid cases	20	
RCA	Measure of Agreement Kappa	4.985	.836
	No of valid cases	20	

H. Chi square test between wall motion assessed by echocardiography and by cine MRI eyeballing method {Table 11.5}

Table 11.5 Wall motion assessed in ECHO * MRI wall motion visual Crosstabulation

		MRI WALL MOTION VISUAL				
		Normal	Mild to Moderate	Severe	Akinesia and Dyskinesia	Total
ECHO wall Motion	0	124	16	16	17	173
	1	36	22	42	48	148
	2	3	2	1	5	11
	3	0	0	0	8	8
Total		163	40	59	78	340

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.065E2[a]	9	.000
Likelihood Ratio	106.529	9	.000
Linear-by-Linear Association	78.126	1	.000
N of Valid Cases	340		

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Measure of Agreement	Kappa	.216	.028	7.717	.000
N of Valid Cases		340			

I. MRI Viability (Delayed enhancement) wall motion abnormality assessed by measurement method {Fig 21.5}

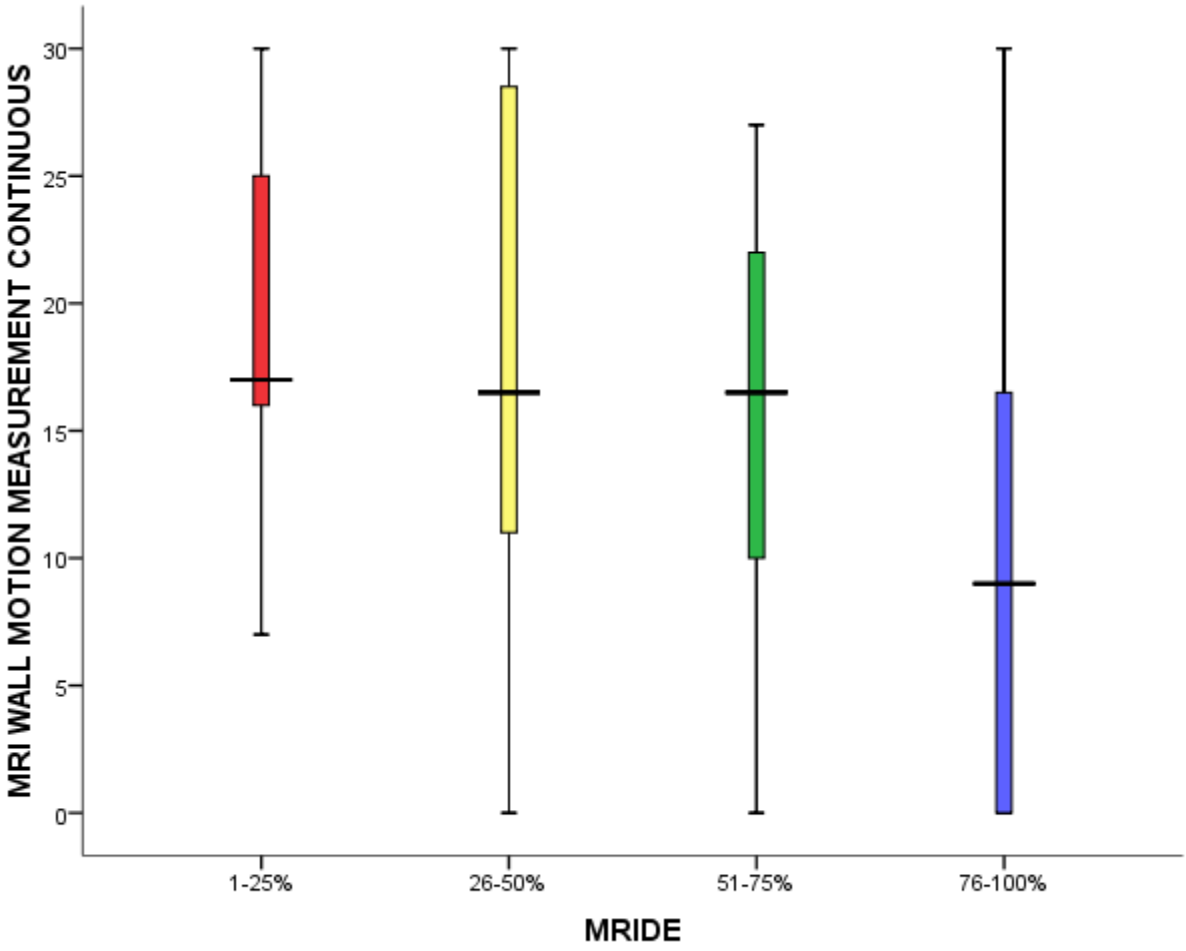

Fig 21. 5 Box and whisker plot showing wall motion assessed by measurement method and extent of delayed enhancement in the myocarcdium

DISCUSSION:

This study was a prospective analytical and descriptive study among patients with symptomatic ischemic heart disease and being evaluated for further management.

Population profile:

A total of 340 myocardial segments from 20 patients who presented with symptomatic ischemic heart disease and required complete evaluation including coronary angiography and cardiac MRI for viability of myocardium were recruited in our study.

The mean age of the study population was 56 years. Average age for males was 57.2 years and the average age for females was 52.6 years. 50% of the patient population was in the age group of 50-60 years. The mean age of our study population is a decade younger than the population suffering from ischemic heart disease in western and other developed nations. This is in keeping with information that we get from current literature. All our patients except two had previous history of angina and were aware of their condition. 85% of our study population was male and the remaining 15% were females. This gender bias in our study can be attributed to male sex seeking medical help more than the female sex due to various reasons such as being the bread earner of the family, being more educated and active etc. The most common comorbidity seen in our study was diabetes mellitus which is present in 55% of our study population.

A total of 340 myocardial segments were assessed in this study. We found 177 segments out of the 340 segments to have dysfunctional myocardial contraction on echocardiography and on cine MRI eyeballing method where as fewer dysfunctional segments were identified by the measurement method. This is because the measurement method was poor in identifying segments that were mild to moderately hypokinetic, the disease being more subtle and thereby making accurate measurement and calculation more difficult.

Of the total of 20 ECG analysed, 18 (%) showed evidence of previous infarct whereas infarct was evident in 100% of the study population on cardiac MRI viability scan.

In the two patient's ECG which showed no direct evidence of previous ischemic insult, the finding in one was a left bundle branch block and the other was non specific. However both patients had significant symptoms and therefore proceeded to have full evaluation. The ECG showing left bundle branch block on evaluation with coronary angiography showed significant disease in left anterior descending artery and right coronary artery and with MRI showed 1-25% of delayed enhancement on post contrast images in the right coronary artery territory.

The ECG showing non specific findings on further evaluation showed minimal disease in the left anterior descending artery however viability scan with MRI showed >75% of delayed enhancement in the left anterior descending artery territory.

Intra class correlation, Pearson's correlation and Bland- Altman plot was generated for the values of ejection fraction measured by echocardiography and MRI short axis cine. The intra class correlation between the two was found to be 55.5% which is statistically significant with p value measuring 0.005. The Pearson's correlation was found to be 60.2% which is statistically significant with a p value of 0.005. The Bland- Altman plot showed the difference between the all ejection fraction measured by echocardiography and MRI to lie within the 95% confidence interval or within 2 standard deviation. The histogram of the difference showed normal distribution curve.

On comparing the wall motion of the myocardium assessed visually on echocardiography and visually on magnetic resonance imaging, we found that they correlate significantly with p value being <.05% in Chi square test. The Kappa agreement between the two was found to be .216 which is statistically significant with p value being <.05%.

On evaluation of the wall motion on cine MRI images by two methods namely by measurement method and by eye balling, a Box and whisker plot was derived and we found that the mild to moderately hypokinetic segments had highest values or score percentage, the severely hypokinetic segments had lower score percentage and those segments with akinesia and dyskinesia showed lowest percentage score some showing score of 0. We also found that by the measurement method for the wall motion, the median value for the mild to moderate , severe and akinesia – dyskinesia set of wall hypokinesia identified by eyeballing was significantly different from one another suggesting that the method is a good test to characterize wall motion into the mentioned groups of wall motion abnormality.

We performed Kappa agreement test to see whether there was significant agreement between the presence of disease in an artery identified on coronary angiography and the corresponding irrigation segment on cardiac MRI. We found no statistically significant agreement between the two groups although we expected 100% agreement. This can be explained by the fact that any degree of disease including minimal or minor disease identified on coronary angiography was recorded as diseased vessel. This vessel occlusion however is not significant yet to cause definite infarction in the myocardium seen as delayed enhancement on viability scan. This finding probably highlights the limitation of cardiac MRI performance in identifying minor coronary artery disease when used in isolation. It also highlights the fact that coronary angiography in conjunction with viability MRI scanning will give the most information that will contribute to decision making and management of the patient.

When chi square test was carried out to analyze the association between the degree of occlusion in a diseased coronary vessel and the degree of delayed hyperenhancement in the corresponding myocardial segment we found statistically significant association only between the left anterior descending artery occlusion and presence of infarct in the myocardium. This means that the association between a diseased left anterior descending and occurrence of infarct in the irrigation territory in the myocardium is statistically significant. However the same could not be said for the left circumflex artery or the right coronary artery in our study.

On analyzing the transmurality of delayed enhancement on cardiac viability scan and the wall motion abnormality scored by measurement of the wall thickness we found that the median value in the various groups were more or less at the same level in the 1-25%, 26-50% and 51-75% groups of delayed enhancement. This means that the measurement method of reporting wall motion abnormality is not able to distinguish between these groups of delayed enhancement. The median value for the 76-100% of wall motion abnormality is significantly different which means that the measurement method for wall motion abnormality can distinctly identify those segments which will show >76% of delayed hyperenhancement from those that may show <76% of delayed enhancement.

Fig 17 a,b,c : 4 chamber, 2 chamber and cine sections, MRI PSIR sequence showing infarct in the myocardium involving >75% of the wall thickness in all segments at the apical level and apex.

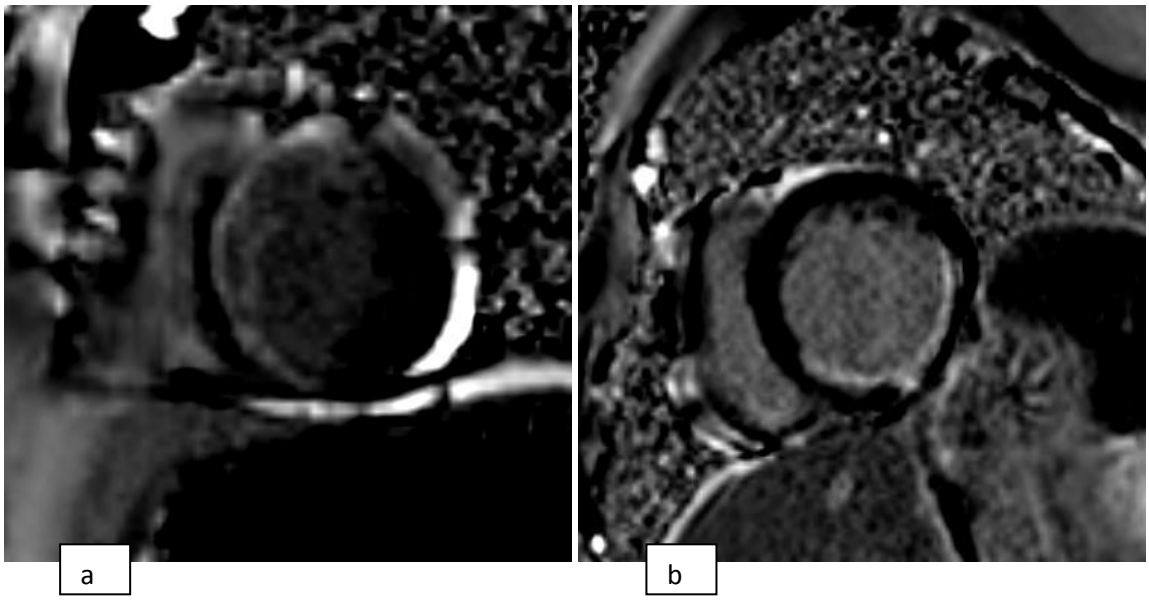

Fig 18 a,b,c : Cine sections, MRI PSIR sequence showing infarct in the myocardium involving 26 t0 50% of the wall thickness in the LAD and RCA territory

CONCLUSIONS

1. Coronary artery disease onset in our study population is a decade earlier than in western or developed countries.

2. Among our study population disease was more common in the male sex.

3. Among the many comorbidities, diabetes mellitus was the most common.

4. In of presence of infarct in the myocardium, cardiac MRI viability better depicts the presence and extent of infarct. It also provides other useful information in the same scan such as presence of wall motion abnormality, left ventricle ejection fraction and accurately depicts the anatomy of the heart and surrounding structures.

5. Same number of segments were identified as contractile dysfunction on echocardiography and eyeballing method on cine MRI images whereas fewer segments were identified by myocardial wall thickness measurement method on MRI images.

6. Wall thickness measurement method is poor in identifying normal myocardial wall motion from mild to moderate wall motion abnormality.

7. Regional wall motion abnormality assessed by eyeballing (MRI) and by myocardial wall thickness measurement (MRI) at end diastole and end systole both are able to distinctly distinguish myocardial segments into three categories: mild to moderate hypokinesia, severe hypokinesia and akinesia to dyskinesia.

8. Wall motion abnormality is not a predictor of presence of infarct. Infarct is present only when there is both wall motion abnormality and delayed enhancement in the same segmental distribution. Delayed hyperenhancement in isolation is a predictor of infarct independently however presence of wall motion abnormality is not.

9. Left ventricular function assessed as ejection fraction by echocardiography and MRI showed significant correlation when assessed by the various statistical methods.

10. Presence of occlusion in coronary vessel does not relate directly to presence of an infarct in the myocardium in most of our cases. Varying degrees of occlusion is seen to cause varying extent of myocardial infarct. This raises the question that other factors such as chronicity or development of collateral blood supply etc play a role in maintaining myocardial viability.

11. Significant association between degree of coronary vessel occlusion and delayed enhancement of the corresponding myocardial segment was seen only in the Left anterior descending artery and its irrigation territory.

12. A protocol consisting of a combination of cardiac MR viability and coronary angiography will give all the information required in management of a patient. MRI is good for follow up studies as there is no radiation exposure and because it is easily comparable.

LIMITATIONS

This study is a hospital based subject selection which comes with its biases and problems and it is difficult to apply these results to the community.

The sample size that could be studied during the given period is less than ideal. This is due to various factors, the most important of which is the expenditure involved with cardiac MRI and shortage of machine time in a tertiary care centre where waiting lists for MR study tend to be very long (~ 5 days).

The small sample size of the study population may not be adequate to bring about statistical significance of the factors that were studied.

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APPENDIX 1

Table 1 Urban prevalence rates of probable CHD per 1000, in males and females From:Heart. 2005 June; 91(6): 719–725. (3)

Study number	Author/date publication	Location and date of fieldwork	Group	Male	Female
Symptomatic and asymptomatic					
1	Gupta 1975	Rohtak, Haryana, dates not given	Adults >30 years	45	28
2	Gupta 1995	Rajasthan, dates not given	Adults >20 years	35	84
3	Gupta 1996	Rajasthan, dates not given	Adult males >20 years	35	NS
4	Singh 1998	Moradabad, dates not given	Adults 25–64 years	90	60
5	Ramachadran 1998	Madras, 1994	Adults >40 years	35	45
6	Mohan 2001	Chennai (formerly Madras), dates not	Adults >20 years	62	93

Study number	Author/date publication	Location and date of fieldwork	Group	Male	Female
		given			
7	Gupta 2002	Rajasthan, dates not given	Adults >20 years		
			Hindu	43	89
			Muslim	7	66
8	Gupta 2002	Rajasthan, dates not given	Adults >20 years	38	72
Asymptomatic					
9	Chadha 1992	Delhi, 1988	Adults 25–64 years		
			Gujeratis	67	31
10	Gopianth 1992	Delhi, 1985–1987	Adults 25–64 years	56	76
11	Singh 1995	Moradabad, dates not given	Adults >49 years	77	56
12	Gopianth 1996	Delhi, 1985–1987	Adults 25–64 years		
			Asymptomatic		
			Hindus	50	61

Study number	Author/date publication	Location and date of fieldwork	Group	Male	Female
			Muslims	95	85
			Sikhs	54	114
			Christians		48
			All	56	76
13	Singh 1997	Rural and urban Moradabad, dates not given	Adults 25–64 years	25	34

Table 2 Rural prevalence rates of probable CHD per 1000 in males and females

From: Heart. 2005 June; 91(6): 719–725. (3)

Study number	Author/date publication	Location and date of fieldwork	Group	Male	Female
Symptomatic and asymptomatic					
1	Dewan 1974	Haryana, dates not given	Adults >30 years	17	13

Study number	Author/date publication	Location and date of fieldwork	Group	Male	Female
2	Gupta 1994	Rajasthan, dates not given	Adults >20 years	28	33
3	Wander 1994	Punjab, dates not given	Adults >30 years	26	38
4	Gupta 1994	Rajasthan, dates not given	Adults >25 years	45	43
5	Gupta 1996	Rajasthan, dates not given	Adults >20 years	28	NS
6	Singh 1997	Moradabad, dates not given	Adults 25–64 years	33	20
7	Gupta 1997	Rajasthan, dates not given	Adults >20 years	28	33
Asymptomatic					
8	Jajoo 1988	Sevagram, dates not given	Adults >30 years	17	34
9	Gupta 1996	Rajasthan, dates not given	Adults >20 years	13	24

Study number	Author/date publication	Location and date of fieldwork	Group	Male	Female
10	Chadha 1997	Delhi, 1984–1987	Adults 25–64 years	6	27
11	Singh 1997	Rural and urban Moradabad, dates not given	Adults 25–64 years	18	11

APPENDIX 2 - Consent form in English, Tamil and Hindi

Information sheet for undergoing cardiac MRI scan

Ischemic heart disease is the leading cause of death in our country. The disease occurs when the blood vessel supplying blood to the muscles of the heart become narrow due to clot formation. There are many cause for clot formation and includes genetics, increasing age, lifestyle, diet etc. Common symptoms of the disease are chest pain and breathlessness on exertion. Investigations like ECG, echocardiography and coronary angiography are done to evaluate the disease and guide the doctor in deciding whether medicines are enough or surgery is required.

We are conducting a study in the department of Radiology, to determine whether cardiac MRI viability will give information similar to the combination of ECG, echocardiography, coronary angiography. The study will involve looking at your ECG, echocardiography and coronary angiography reports and comparing it with the report of the cardiac MRI viability.

Participation is voluntary and if you agree to take part in the study,

- You will not be charged for the cost of the cardiac MRI viability
- You will have to answer few questions and undergo basic general examination to ensure that there are no contraindications to have MRI scan
- You will need to have an injection in your hand for contrast agent to do the scan.
- The MRI scan will last 30-40 minutes during which you will be instructed to hold your breath for a few seconds. You will be coached to hold your breath the correct way before you enter the scan room.
- The machine may make a lotof noise during the scanning however this is normal. We will provide you with ear muffs if you require them.
- There are no known side effects to undergoing the scan itself.
- There is a very small risk that you may be allergic to the contrast agent used.
- The contrast agent can cause renal disease in few patients who already have poor kidney function.

With our study we hope to gain more knowledge and understanding in the role of cardiac MRI viability in the management of ischemic heart disease. This will help in improvement in the way we take care of our patients.

I have read and understood what the study involves and I give consent to undergo cardiac MRI viability scan for myself / relative. I also give consent to viewing of my the reports of my ECG , echocardiography and coronary angiography.
I am aware that participation in the study is voluntary and that I may withdraw from the study at any time.

Name Hospital number

Signature of patient

Or

Signature of relative giving consent Date:

தகவல் தாள்

இருதய நோய் இந்தியரை மற்றோரைவிட இளவயதில் தாக்குகிறது எனவே இதை முன்பே கண்டறிந்து சிக்ச்கை பெறுவது அவசியம்

இருதய MRI ஸ்கேன் மூலம் இவ்வியதியை கண்டறிந்து, எவற்கு எத்தகைய சிகிச்சை வேண்டும் என்று அறிவதுடன் சிகிச்சையினால் எவ்வளவு முன்னேற்றம் அடைய வாய்ப்புகள் உள்ளன என்றும் அறியகூடம்

இவ்வாராய்ச்சியின் மூலம் இருதய MRI ஸ்கேனிற்கும் இருதயத்திற்காய் செய்யப்படும் வேறு பரிசோதனைகளுக்கும் எவ்வளவு ஒற்றுமையுள்ளதென்று அறிய கூடும் இதனை அறிந்தால் இந்த பரிசோதனை யாருக்கு பயன்படும் என்று தீர்மானிக்க உதவும் இதனால் தேவையுள்ளோர் இந்த வசதியை உபயோகித்து பயனடைய உதவும்.

ஒப்பந்த படிவம்

நான் இந்த ஆராய்ச்சியை குறித்து புரிந்து கொண்டேன், என் மீதோ என் உறவினர் மீதோ இந்த பரிசோதனையை நடப்பிக்க சம்மதிக்கிறேன்
இந்த ஆராய்ச்சிக்கா என் மற்ற பரிசோதனை விளைவுகளையும் உபயோகிக்க சம்மதிக்கிறேன்

இந்த ஆராய்ச்சியில் பங்கேற்பது தன்னாற்வமானதென்றும் எப்பொழுதும் நான் இதிலிருந்து விலகிக் கொள்ளலாம் என்றும் அறிகிறேன்.

பெயர் மருத்துவமனை எண்

தொலைபேசி எண்

முகவரி

கையொப்பம் தேதி:

सूचना पत्रीका

अकास्मित हृदय रोग भारत में होने वाली मौतों का एक प्रमुख कारण है। यह रोग छोटी आयु समूहों को प्रभावित करता है। इसलिए इसका उचित उपचार बहुत जरूरी है। यदि उपचार न हो तो मरीज की मौत हो सकती है।

अकास्मित हृदय रोग का स्थायीकरण निदान और उन लोगो को जिनको शल्य चिकित्सा की प्रक्रिया आवश्यकता है, इनकी पुष्टिकरण हमारे संस्था में ई सी जी, इको तथा कोरोनरी एंजियोग्राफी के रूप में जाँच प्रक्रिया की जाती है।

हृदय इमेजिन इतना उन्नत हो गया है कि इससे केवल निदान के लिए बल्कि हृदय की कार्यात्मक भविष्य में कितना होगा उसका भी अध्ययन किया जा सकता है।

इस अध्ययन से हम हृदय राम आर आई (mRI) की तुलना अन्य हृदय जाँच जिसेकी ई.सी.जी (ECG) और इको (ECHO) और कोरोनरी एंजियोग्राफी से कर सकते है एवं सहसंबंधी बना सकते है। इससे हमें

अकास्मित हृदय रोग के बारे में अच्छी समझ एवं राम आर आई (mRI) से इस रोग के बारे में अच्छा मूल्यांकन किया जा सकता है। मरीजों को इस निदान से बेहतर चिकित्सा उपलब्ध हो सकती है।

सहमति फॉरम

मुझे इस अध्ययन के बारे में अच्छि समझ है। मैं अपनी सहमति देता हूँ/ इस अध्ययन को अपने संबंधी पर करने के लिए सहमति देता हूँ। मुझे पता है कि इस अध्ययन में मेरी भागीदारी स्वैच्छिक है और इसे अपना नाम किसी भी समय वापस ले सकता हूँ।

नाम :

अस्पताल नंबर :

टेलीफोन नंबर :

पता

हस्ताक्षर

दिनांक

APPENDIX 3 - Data collection form

I) Patient characteristics/demography:

Study number: _____

Name::_____

HOSPITAL NUMBER:

Age;_____yrs Sex: ___ (M/F)

Address:_____

_____

_____

_____

_____

II) Clinical Parameters:

i) No of years of disease: _____ months /years
ii) NYHA Class:_____
iii) Comorbidities:_____
iv) Past history of heart surgery:_____

I) Investigation:

SL NO.	INVESTIGATION	DATE:	Involved wall on ECG	Regional Wall motion abnormality (ECHO and CMRI)	Involved vessel (Coronary angiogram)	Percentage of occlusion (coronary angiogram)	Extent of transmural enhancement on CMRI (%)
1.	ECG						
2	ECHO						
3.	Coronary angiography						
4.	Cardiac MR viability study						

APPENDIX 4

ECG REPORT

Name: _____Age:_____

Hospital no._____

Indication:_____

HEART RATE:_____

RHYTHM:_____

QRS axis:_____

P wave morphology:_____

PR interval:_____

QRS duration / morphology:___

Comment on ST segment:_____

T wave abnormalities:_____

Any other significant findings:_

CONCLUSION:

APPENDIX 5

ECHOCARDIOGRAPHY REPORT

AORTIC VALVE: Normal / _____ ; Trileaflet / bileaflet

MITRAL VALVE: Normal / _____

PULMONIC VALVE: Normal /_____

TRICUSPID VALVE: Normal /_____

LEFT ATRIUM: Normal / _____

RIGHT ATRIUM: Normal /_____

PULMONARY ARTERY: Normal /_____

ATRIAL SEPTUM: Normal /_____

LEFT VENTRICLE:

Size: _____

Function: _____

Ejection fraction:____

THE LEFT VENTRICULAR WALL MOTION: Normal/ mild or moderate hypokinesia/ severe / hypokinesia / akinesia / dyskinesia _____

PERICARDIUM: Normal / _____ AORTA: Normal /_____

CONCLUSION:

APPENDIX 6

CORONARY ANGIOGRAPHY REPORT

PROCEDURE MEDICATIONS:

1. Tab Valium 5 mg before procedure 2. Inj Hydrocortisone if required (contrast allergy)

DESCRIPTION OF PROCEDURE:

APPROACH: Left heart catheterization via right radial artery / any other__

ACCESS METHOD: Percutaneous needle puncture

FINDINGS/INTERVENTIONS:

LEFT VENTRICULOGRAPHY (If applicable): Not applicable / Findings:_____

LEFT MAIN CORONARY ARTERY: normal / _____

Intervention (done or advised):

LEFT ANTERIOR DESCENDING ARTERY: normal / _____

Intervention (done or advised):

LEFT CIRCUMFLEX ARTERY: normal / _____

Intervention (done or advised) :

RIGHT CORONARY ARTERY: normal / _____

Intervention (done or advised) :

COMPLICATIONS:

There were no complications during the procedure.

IMPRESSION:

RECOMMENDATION:

1. Clopidogrel (Plavix) 75 mg PO daily for 1 year.
2. Aggressive risk factor modification of tobacco abuse, hyperlipidemia and hypertension.
3. _____

APPENDIX 7

CARDIAC MRI REPORT

Levo cardia
Situs solitus
Normal atrio-ventricular concordance
Normal ventriculo-atrial concordance
Normal veno- atrial concordance
No obvious septal defects

VALVES_____

CHAMBER MORPHOLOGY_____

PERFUSION SCAN_____

VIABILITY:

LV REGIONAL WALL MOTION
(0=normokinetic; 1=mild/moderate hypokinesia; 2=severe hypokinesia; 3=akinetic; 4=dyskinetic)

BASE
Anterior:
Anteroseptal:
Inferoseptal:
Inferior:
Inferolateral:
Anterolateral:

MID
Anterior:
Anteroseptal:
Inferoseptal:
Inferior:
Inferolateral:
Anterolateral:

APICAL
Anterior:
Inferior:
Septal:
Lateral:

APEX:

LV MYOCARDIAL ENHANCEMENT
(0=none; 1=1-25%; 2=26-50%; 3=51-75%; 4=76-100%)

BASE
Anterior:
Anteroseptal:
Inferoseptal:
Inferior:
Inferolateral:
Anterolateral:

MID
Anterior:
Anteroseptal:
Inferoseptal:
Inferior:
Inferolateral:
Anterolateral:

APICAL
Anterior:
Inferior:
Septal:
Lateral:

APEX:

PAPILLARY MUSCLES:

PERICARDIUM:

EXTRA CARDIAC: